EFFECTS OF AUTOMOTIVE EMISSIONS

by

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FOR

Environmental Protection Service
Environment Canada

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IP-7
January, 1984

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ABBREVIATIONS, ACRONYMS, AND SYMBOLS

Å	Ångstrom (10^{-10} meter)
ARI	Acute respiratory illness
AS	Asthma
ASTM	American Society for Testing and Materials
atm	One atmosphere, a unit of pressure
avg	Average
BS	British smoke
$^{\circ}C$	Degrees Celsius (centigrade)
CB	Chronic bronchitis
CE	Controlled environment
cm	Centimetre
CNS	Central nervous system; the brain and spinal cord
CO	Carbon monoxide
CO_2	Carbon dioxide
COH	Coefficient of haze
COHb	Carboxyhaemoglobin
COPO	Chronic obstructive pulmonary disease
CRD	Chronic respiratory disease
CVO	Cardiovascular disease
d	Day
DL_{CO}	Diffusion capacity of the lung for carbon monoxide
D = CT	Dose equals concentration multiplied by time
EEC	European Economic Community
EKG	Electrocardiogram
EPA	U.S. Environmental Protection Agency
$^{\circ}F$	Degrees Fahrenheit
FEF	Forced expiratory flow
$FEF_{25\%-75\%}$	FEF between 25 and 75% of FVC
FEV	Forced expiratory volume
FMC	Fine particle mass concentration
FVC	Forced vital capacity
GH	Greenhouse

ABBREVIATIONS, ACRONYMS, AND SYMBOLS (CONT'D)

h	Hour
H	Hydrogen
ha	Hectare
Hb	Haemoglobin
HbO_2	Oxyhaemoglobin
HC	Hydrocarbon
HNO_2	Nitrous acid (also HONO)
HNO_3	Nitric acid (also $HONO_2$)
HO	Hydroxyl free radical (also OH)
hr	Hour
H_2SO_4	Sulphuric acid
IARC	International Agency for Research on Cancer
IFR	Instrument Flight Rules
in	Inch
inc/prev	Incidence/prevalence
k	Rate constant or dissociation constants
kg	Kilograms
km	Kilometre
l	Litre (also ℓ)
LRD	Lower respiratory disease
m	Metre
max	Maximum
Meq/L	Microequivalent per litre
$\mu g/m^3$	Micrograms per cubic metre
mg/m^3	Milligrams per cubic metre
ml	Millilitre
mo	Month
N	Nitrogen
NA	Not applicable
NAAQS	National Ambient Air Quality Standard
NaCl	Sodium chloride; common table salt

ABBREVIATIONS, ACRONYMS, AND SYMBOLS (CONT'D)

NAQO	National Air Quality Objective
NAS	National Academy of Sciences
NCLAN	National Crop Loss Assessment Network
ng	Nanogram
NH_4	Ammonium ion or radical
NO_x	Nitrogen oxides
N_2O	Nitrous oxide
NO_2	Nitrogen dioxide
N_2O_4	Dinitrogen tetroxide
NR	Natural rubber
NRCC	National Research Council of Canada
O	Atomic oxygen
OAQPS	Office of Air Quality Planning and Standards (EPA)
O_3	Ozone
OH	Hydroxyl group
$PaCO_2$	Arterial partial pressure of carbon dioxide
PAH	Polycyclic aromatic hydrocarbon
PAN	Peroxyacetyl nitrate
PaO_2	Arterial partial pressure of oxygen
pH	Log of the reciprocal of the hydrogen ion
PO_2	Partial oxygen pressure
ppb	Parts per billion
pphm	Parts per hundred million
ppm	Parts per million
ppt	parts per trillion
R_{aw}	Airway resistance
RSP	Respirable suspended particulate
SBR	Styrene-Butadiene Rubber
SD	Standard deviation
SN	Suspended nitrates
SO_2	Sulphur dioxide

ABBREVIATIONS, ACRONYMS, AND SYMBOLS (CONT'D)

SR_{aw}	Specific airway resistance
SS	Suspended sulphates
TLC	Total lung capacity
TLV	Threshold limit value
TSP	Total suspended particulate
TWA	Time-weighted average
VFR	Visual flight rules
V_{max}	Maximum expiratory flow rate
WHO	World Health Organization
wk	Week
yr	Year
Mg	Microgram
Mm	Micrometre
>	Greater than
<	Less than
∿	Approximately

CONVERSION OF CONCENTRATIONS

The following conversion factors have been used in the present document (based on ambient conditions of 25°C, 1 atm):

Carbon monoxide (CO)	1 ppm	=	1.145 $\mathrm{mg/m^3}$ = 1145 $\mu\mathrm{g/m^3}$
Nitric oxide (NO)	1 ppm	=	1.230 $\mathrm{mg/m^3}$ = 1230 $\mu\mathrm{g/m^3}$
Nitrogen dioxide (NO_2)	1 ppm	=	1.880 $\mathrm{mg/m^3}$ = 1880 $\mu\mathrm{g/m^3}$
	1 ppb	=	1880 $\mathrm{mg/m^3}$ = 1.880 $\mu\mathrm{g/m^3}$
Ozone (O_3)	1 ppm	=	2.000 $\mathrm{mg/m^3}$ = 2000 $\mu\mathrm{g/m^3}$
	1 ppb	=	2000 $\mathrm{mg/m^3}$ = 2.000 $\mu\mathrm{g/m^3}$
Peroxyacetylnitrate (PAN)	1 ppm	=	5.000 $\mathrm{mg/m^3}$ = 5000 $\mu\mathrm{g/m^3}$
	1 ppb	=	2000 $\mathrm{mg/m^3}$ = 5.000 $\mu\mathrm{g/m^3}$
Sulphur dioxide (SO_2)	1 ppm	=	2.600 $\mathrm{mg/m^3}$ = 2600 $\mu\mathrm{g/m^3}$
	1 ppb	=	2600 $\mathrm{mg/m^3}$ = 2.600 $\mu\mathrm{g/m^3}$

1. EXECUTIVE SUMMARY

1.1 Introduction

A review of the effects of automotive emissions was carried out for Environment Canada as part of a Socio-Economic Impact Analysis (SEIA) in relation to proposed revision of Canadian Automobile Emission Control Standards. This document investigated the following pollutants related to automobile emissions: carbon monoxide, nitrogen oxides, photochemical oxidants, hydrocarbons, and diesel exhaust particulates. The effect of the nitrate component of acid precipitation, formed from nitrogen oxides, was also reviewed.

The rationale for establishing ambient air quality objectives and guides was investigated to establish the areas of concern for adverse effects. This procedure for setting air quality objectives provides an interpretation of what is considered a non-acceptable effect and of what is a "safe" level to provide protection from these effects. The approach used in defining or promulgating a "safe" level is important because rarely is there sufficient scientific information about a pollutant to provide this distinction. In these cases a margin of safety is usually provided to account for uncertainties.

Adverse effects documented in this report include:

1. Health effects of carbon monoxide, nitrogen oxides, ozone, diesel exhaust particulates and hydrocarbons.
2. Terrestrial ecosystem effects, particularly crop damage.
3. Aquatic ecosystem effects, especially fisheries (acid rain).
4. Material damage.
5. Visibility reduction by particulate and gaseous emissions and photochemical products.

The documentation of acid rain formation from automotive air pollution (particularly NO_x) and its environmental effects also constitute a part of this document. In a similar manner, the formation of photochemical oxidants is reviewed to provide a consistent approach of summarizing the effects (both direct and indirect) of automotive air pollution.

The cost-benefit approach being used by the SEIA requires rather precise dose-response functions for each effect. Assessment of the present information base shows that major gaps in knowledge exist for each of the pollutants covered in this report. Additionally, there can be disagreement on what constitutes an adverse effect. Whenever possible, and within the limitations of current knowledge, dose-response functions have been provided as input to the SEIA.

This report relies heavily upon air quality criteria documents by Environment Canada, the United States Environmental Protection Agency, the World Health Organization and other agencies. No attempt was made to comment on all of the primary documents referred to. Rather, original works or reviews believed to be most significant are included plus any other significant published work reported since the dates of the criteria documents.

1.2 Air Quality Standards

Ambient air quality standards or objectives may vary from country to country and within a country over the course of time as new knowledge is acquired. The long-term goal is generally to protect against adverse effects on human health. Consideration is also given to adverse effects on the environment such as vegetation damage, materials damage, and visibility impairment. The concept of a standard to protect receptors from significant harm is a statistical one, based on consideration of an aggregate of individual cases of significant harm. The adoption of a standard may not confer protection on every individual. The following provides a brief synopsis of air quality standards for carbon monoxide, nitrogen dioxide, hydrocarbons, and photochemical oxidants. No air quality standards have been promulgated in Canada for diesel exhaust particulates or acid deposition.

Carbon Monoxide (Section 3.4.1)

The generally recognized 'safe' carboxyhaemoglobin (COHb) levels in blood have decreased over the last decade, resulting in lower existing guides and proposed standards. There is also more recognition of the rapid accumulation of COHb by exercising individuals. The current CO maximum acceptable limit of 34 mg/m^3 (30 ppm) for 1-hour, recommended in 1971, is 6 mg/m^3 (5 ppm) higher than the more recent World Health Organization (WHO) recommendations and the proposed United States standard. Similarly, the Canadian acceptable limit for 8-hour exposure is higher than the existing United States standard and the 1979 WHO recommendation. Only the Japanese 8-hour standard which was set in 1971 is higher than the Canadian counterpart.

All of the CO standards set or recommended are directed at the protection of vulnerable groups such as cardiac patients and anaemics from adverse effects. In the United States approach, there is the concept of providing a margin of safety for sensitive groups visiting high altitude locations (e.g. a cardiac patient visitor from New York to Denver) and the fetus. There is also uncertainty in model relationships between CO exposure and COHb blood levels. Currently, one of the aspects being explored by the U.S. EPA and the Canada Subcommittee on National Air Quality Objectives is the sensitivity of the Coburn Model predictions of COHb levels associated with variable rather than static CO concentrations and with different physiological parameters.

Nitrogen Dioxide (Section 3.4.2)

The long-term standards for NO_2 for all countries are in the vicinity of 100 $\mu g/m^3$ (0.05 ppm) NO_2 based on protecting the health of sensitive groups of the population. Of the countries with short-term standards (daily or 1-hour averages), the primary criteria have been health effects, but consideration has also been given to the role of NO$_2$ in the formation of photochemical pollution (Japan and West Germany), plant damage (Canada), and sensory perception (Canada). The scientific criteria available for a long-term standard based on health effects are very tenuous and the World Health Organization as of 1977 found insufficient information to recommend an air quality guide. The U.S. EPA review of scientific criteria also reached this conclusion, but since an annual standard was in place, it was felt that this level could be used as a surrogate for protection against short-term peaks of NO$_2$. An appreciable safety factor of 3 to 5 was incorporated into the 1-hour WHO health guideline because of the high biological activity of the NO$_2$.

Hydrocarbons (Section 3.4.3)

There are no air quality objectives for hydrocarbons in Canada. In 1983, the United States revoked its non-methane hydrocarbons standards of 160 μg/m^3 (0.24 ppm) 3-hour average from 6 to 9 a.m. The past standard had been unique in that the levels set were not based on direct health or welfare effects of hydrocarbons, but was intended to provide a means of attaining the ozone standard.

Photochemical Oxidants (Section 3.4.4)

The chronological history of setting of standards and recommendation of guidelines results in conflicting trends. The United States relaxed their 1-hour standard from 160 μg/m^3 (80 ppb) to 240 g/m^3 (120 ppb) in 1979, while the WHO in the same year reduced their guidelines for protection of human health from the range of μ 200 250 μg/m^3 (100-125 ppb) to 100-200 μg/m^3 (50-100 ppb). The Canadian maximum acceptable objective is 160 μg/m^3 (80 ppb) for 1-hour average. These differing interpretations are essentially based on the same set of scientific criteria, although the WHO tends to use animal data more than the United States in support of their recommendations.

Overall, ozone standards have been set primarily to protect public health. In Canada, vegetation damage was also considered in establishing maximum acceptable objectives. The province of Ontario's air quality criteria for ozone is primarily based on avoidance of vegetation damage.

1.3 Effects of Automotive Emissions

Air quality criteria are observed effects or responses of one or several atmospheric substances on a defined receptor or population under specified conditions. These criteria are used as a basis of standard-setting, and in cases where information is complete are synonymous with dose-response relations. The following provides a brief summary of the effects of automotive air pollutants on receptors or populations.

1.3.1 Health

Carbon monoxide (CO) has deleterious effects on cardiac function which is of greatest relevance to individuals with cardiovascular disease. Carbon monoxide also has an adverse effect on work performance and on central nervous system functions. Impairment of vigilance or performance of complex tasks can occur at low levels of CO exposure which may be of significance to the general population, especially with respect to its possible effect on driving skills. No such effect has, however, been clearly documented. Pregnant women, their fetuses, and newborn babies, have increased susceptibility to carbon monoxide toxicity, but precise quantification of increased risk for this group is not yet possible.

Exposure to nitrogen dioxide (NO_2) affects sensory perception and causes irritation to the mucous membranes of the respiratory tract. The adverse effect of NO_2 on lung function may be of particular relevance to asthmatics. It also appears that NO_2 increases susceptibility to infection, possibly accounting for an increased incidence of respiratory disease, most notably in children.

Exposure to ozone may induce abnormalities in various organs throughout the body. Again, the respiratory system is the site of greatest damage. Ozone impairs lung function, with the magnitude of the effect dependent on a variety of factors, including individual susceptibility and level of exercise. Decreased performance, exacerbation of asthma, increased susceptibility to infection and excess respiratory disease hospital admissions have all been linked to ozone exposure.

Diesel exhaust is known to contain a high concentration of particulates, with the latter having been linked to increased mortality among elderly individuals and patients with respiratory disease (chronic bronchitis, emphysema, etc). Exposure to total suspended particulates is also known to cause exacerbation of disease in such patients, and studies have linked the frequence of asthma attacks and incidence of acute respiratory disease in children with exposure to particulates. Diesel exhaust emissions are composed primarily of very fine particles and it is not known to what extent these fine particles contribute to respiratory disease. The major health concern regarding diesel emissions is its potential to be cancer-causing. At present, no convincing evidence exists to substantiate, let alone quantify, this risk to humans.

Hydrocarbons as a class provide little direct health effect at ambient levels. Some hydrocarbons, however, can be hazardous at levels approximating those found in the ambient air. Benzene, for example, known to be capable of producing serious blood disorders, including leukemia, may be of concern in some specific settings. Aldehydes, such as formaldehyde, which may be formed from hydrocarbons emitted in automobile exhaust, may be irritating to the mucous membranes or cause asthmatic attacks in sensitive individuals. Quantification of this effect is not possible at this time.

Acid precipitation may also indirectly affect health by increasing the risk of mercury poisoning, lead poisoning or intoxication from other elements in some areas and populations.

1.3.2 Terrestrial Systems

Studies of air pollutant effects on terrestrial systems have concentrated mainly on vegetation. Visible leaf injury is the most readily detected and most frequently reported symptom of exposure to NO_2, and has been documented in many reports of damage to economic crops. Studies have shown, however, that declines in plant growth and yield can occur without such visible symptoms.

Exposure to short-term peaks in NO_2 may cause as much or more damage to vegetation than does continuing exposure over a growing season. Growth and yield reductions may take place with minimal or no foliar injury while it is also possible to detect extensive foliar injury with no significant effects on crop yields.

Studies on combined exposures to NO_2 and SO_2 have found that the NO_2 injury threshold can be significantly reduced in the presence of SO_2. Responses to mixtures are related to concentration ratios, sequence of exposure and other variables. Ambient concentrations in some areas of North America are within ranges at which observable injuries have occurred.

The available data suggest that ozone (O_3) is the most pervasive and economically damaging of the photochemical oxidants. Ozone effects on plants include metabolic alterations, reduced primary production, and subsequent changes in the partitioning of photosynthate and related reductions in biomass and/or yield. Other resulting effects include those that relate to plant quality, such as foliar injury or altered nutritional composition of crops. Studies note that while foliar injury is a useful indicator of the presence of photochemical oxidants, it may be caused by other stresses and may not always be a reliable predictor of effects of oxidants on growth, yield or quality. Current evidence suggests that ozone in ambient air in many areas of North America is sufficient to reduce the growth and yield of vegetation, and that sensitive crops are significantly affected by a 7-hour midday seasonal average O_3 concentration of 80 to 200 $\mu g/m^3$ (0.04 to 0.10 ppm).

There is evidence to suggest that major portions of temperate forest ecosystems are undergoing perturbations related to oxidants, resulting in significant forest changes. Decreases in production and diversity are evident, with early and mid-successional forests being particularly endangered. Long-term continual stresses are thought to decrease total foliar cover and species richness and increase dominance by oxidant-tolerant species. Gradual and subtle changes over time in forest metabolism, growth and composition over wide areas of the temperate zone are thought by researchers to be much more important than the more dramatic destruction of forests in the immediate vicinity of point sources of oxidants over short periods.

Nitrogen inputs from acid precipitation are probably insignificant as a nutrient source relative to agricultural crop requirements, but may supply as much as 40% of forest nitrogen requirements. There is currently little certain evidence that existing acid precipitation levels are having deleterious effects upon terrestrial vegetation, but it may affect the productivity of forest and crop plants by direct impaction, changes in soil structure and processes, or leaching of minerals from watersheds. Foliar injury due to acidic deposition has not been documented in the field, but treatments in controlled conditions with simulated acidic precipitation have resulted in foliar injury, growth reductions and growth stimulations in some cases. Too few studies have been completed to assess accurately the terrestrial effects of acidic precipitation.

1.3.3 Aquatic Systems

Acidic sulphur inputs exceed acidic nitrogen inputs over eastern North America on an annual basis and the net yield of these anions to streams and lakes is predominantly sulphate on an annual basis. Researchers have concluded that since nitrate reaches surface waters in small amounts relative to its loadings on an annual basis and does not accumulate in surface waters, its influence on long-term surface water acidification is less than sulphate.

Nitric acid may play a more significant role in short-term acidification associated with spring melt of nitrate-containing snowpack. Thus, nitrate might be important on an episodic basis by adding to the existing pH depression caused by sulphate. Studies to date of this phenomenon have found that sulphate concentrations still exceeded nitrate on an equivalent basis, even during spring runoff.

The evidence and conclusions of published sources lead to the conclusion that for surface water systems, most of the increases in acidity observed was due to the changes noted in sulphate concentration attributed to sulphate and sulphuric acid deposition. Both sulphuric and nitric acids contribute acidity to surface waters during periods associated with pH depressions and fish stress. There is no strong evidence at present for anticipating any appreciable reduction in long-term lake or stream acidification from a reduction in nitrate inputs.

Atmospheric nitrogen may supply a significant proportion of loadings to the Great Lakes and other Canadian waters. In most lakes, eutrophication is largely limited by phosphorus availability, so that atmospheric nitrogen inputs are unlikely to be a major contributer to lake enrichment. Coastal and estuarine ecosystems, and currently eutrophied lakes are exceptions where increased nitrogen supplies may contribute to stimulated aquatic production.

1.3.4 Man-Made Materials

Damage to some man-made materials by ozone and nitrogen dioxide has been documented at levels experienced in the ambient atmosphere. However, caution must still be used in applying documentation of material damage in chamber studies to real life exposures.

Cellulosic fibres (cotton and viscose rayon) dyed with direct dyes, vat dyes, and fibre reactive dyes, suffer severe fading on chamber exposures to 940 $\mu g/m^3$ (0.5 ppm) NO_2 with high humidity (90%) and high temperatures (30°C). Significant fading is observed on 12 weeks exposure to 100 $\mu g/m^3$ (0.05 ppm) under the same high humidity and temperature conditions. Acid dyes on nylon fade on exposure to NO_2 at levels of 200 $\mu g/m^3$ (0.1 ppm) under the same environmental conditions. Yellowing of white fabrics for polyurethane segmented fibres, rubberized cotton, optically brightened acetate, and nylon have been documented in chamber studies using 400 $\mu g/m^3$ (0.2 ppm) NO_2 for exposure of 8-hours. Nitrates have been implicated in the cracking of wires made of nickel brass alloy that are used in telephone equipment, but no other evidence exists for metal corrosion. Since nitrate salts are more hygroscopic than chloride or sulphate salts, nitrates may lower the threshold requirements for wet metal corrosion.

The only quantified effect of ozone on materials is for tire products. Stressed natural rubber cracks at ozone concentration as low as 40 $\mu g/m^3$ (20 ppb). Tires are protected from ozone degradation by the addition of antiozonants which inhibit ozone reactions. There are effects of ozone on textile dye fading under select conditions but this can not be readily generalized to the urban environment. All effects occurred at high temperatures and high relative humidites. Some recent work has indicated there may be a significant association between the erosion of latex paint and relative humidity and ozone but further work is required to provide a definitive dose-response damage function. No evidence currently exists to substantiate any adverse effects on structural materials by ozone, nitrogen dioxide, or nitrates.

1.3.5 Visibility

Visibility is recognized as being important to perceived air quality by people in general and by government agencies. One of the criteria used to establish the Canadian maximum air quality objective for suspended particulates was protection against visibility impairment. Recently both the U.S. Environmental Protection Agency and the Canadian National Research Council have recommended that standards (Objectives) be established for fine particles with visibility as a criteria on the basis of the relatively well defined relationship between visual range and fine particle mass. These fine particles in many cases are photochemical products of gaseous emissions. The basis for estimating visual-range from fine particle mass concentration is reasonably well known. Semi-empirical relationships may be used as an indicator of the relative importance of the various chemical species present in the fine particle mode to visibility impairment. The importance of sulphates in visibility reduction in eastern North America is firmly established, while nitrates, organic compounds, and carbon (soot) are more site dependent.

The recent United States-Canada Memorandum of Intent document on long-range transport concludes that "available data suggest that nitrates exist predominantly in the vapour phase and are for the most part of little consequence to visibility in eastern North America."

The contribution of nitrogen dioxide to reduction of visual range is small, but it may cause a brownish haze in some circumstances; however, in the majority of cases, brownish haze has been attributed to particle scattering.

1.3.6. Indirect Effects

Indirect effects include the formation of a secondary reaction product which itself has a direct effect as summarized in the previous five areas of human concern. These indirect effects include the formation of photochemical oxidants from primary emissions of hydrocarbons and nitrogen oxides in the presence of sunlight and the formation of nitrates and nitric acid from emissions of nitrogen oxides. The end products all have known or suspected adverse effects on various receptors.

The quantitative relationship between precursors (NO_x and hydrocarbons) and end products (O_3, HNO_3) is very complicated and not completely understood.

The role of NO_x emissions in formation of photochemical oxidants is a matter of controversy. Some studies suggest that decreasing present NO_x levels will promote formation of ozone.

All of the conversion processes for acid precipitation require oxidizing substances such as hydrogen peroxide, ozone, and hydroxyl radicals. The production of these compounds is directly related to the reactive hydrocarbon pollutant class. The conversions of air pollutants to acid aerosols all occur at different rates, depending on the relative and absolute amounts of sulphur dioxide, nitrogen oxides, hydrocarbons, and fine particles present in emissions. Meteorological conditions also greatly influence overall conversions. The acid conversion process on an event basis (short-time period) may be characterized by SO_2 and NO_x competition for oxidants and inhibition of one acid's formation by the presence of the other compound. However, over a longtime and over a large regional scale the reactions may possibly be considered linear. In other words, the concentration of the acid formed may be directly proportional to the emission amount of its precursor.

RÉSUMÉ À L'INTENTION DE LA DIRECTION: RÉPERCUSSIONS NUISIBLES DES ÉMISSIONS DES VÉHICULES AUTOMOBILES*

préparé par

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1 Introduction

Une étude des émissions de véhicules automobiles et de leurs répercussions nuisibles a été effectuée pour Environnement Canada dans le cadre de l'Analyse des incidences socio-économiques (AISE) de normes proposées limitant les émissions des véhicules automobiles neufs. Au cours de cette étude des recherches approfondies ont été faites sur les polluants émis par les véhicules automobiles, nommément le monoxyde de carbone, les oxydes d'azote, les oxydants photochimiques, les hydrocarbures et les particules émises par les systèmes d'échappement de moteurs diesel. Les répercussions des composants nitriques des précipitations acides formés à partir des oxydes d'azote, ont également été étudiées.

La philosophie sur laquelle repose les normes et les lignes directrices a été le fruit d'une réflexion approfondie dans le but de cerner les principales préoccupations que soulèvent les effets nuisibles sur la qualité de l'air. L'élaboration des normes nécessite un jugement sûr pour faire la distinction entre un effet inacceptable et une concentration maximale admissible, c'est-à-dire celle qui assure une protection contre les répercussions nuisibles. La méthode utilisée pour définir ou imposer une concentration maximale admissible est importante parce qu'on dispose rarement d'une information scientifique suffisante sur un polluant pour établir cette distinction. Dans ces cas, une marge de sécurité est habituellement incorporée pour tenir compte des incertitudes.

Les polluants susmentionnés ont des répercussions nuisibles dans les cinq domaines suivants:

1) La santé, à cause du monoxyde de carbone, des oxydes d'azote, de l'ozone et des particules émises par les systèmes d'échappement de moteurs diesel.
2) L'écosystème terrestre, en particulier les dommages causés aux récoltes.
3) L'écosystème aquatique, spécialement sur les pêches (précipitations acides).
4) Les dommages matériels.

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* MAS, contrat n° KE145-2-0692, octobre 1983

5) **La réduction de la visibilité par les émissions particulaires et gazeuses et les produits photochimiques.**

De plus, le présent document traite de la formation des précipitations acides provenant des émissions de véhicules automobiles (particulièrement NO_x) et de ses répercussions. On y expose aussi les résultats d'une étude sur la formation des oxydants photochimiques qui permettront d'élaborer une méthode logique visant à identifier sommairement les répercussions nuisibles, directes et indirectes, des émissions des véhicules automobiles.

La méthode d'analyse des coûts et des rendements utilisée pour l'AISE exige que les fonctions "dose-réponse" soient précises pour chaque répercussion nuisible. L'évaluation de la base d'information actuelle montre qu'il existe d'importants manques de connaissances sur chacun des polluants étudiés dans le présent rapport. De plus, il peut y avoir désaccord sur ce qui constitue une répercussion nuisible. Lorsque c'est possible et dans les limites des connaissances actuelles, les fonctions "dose-réponse" ont été incorporées dans les données utilisées pour faire l'évaluation de l'analyse des incidences socio-économiques.

Le présent rapport est basé principalement sur les documents relatifs aux critères de la qualité de l'air élaborés par Environnement Canada, l'Environmental Protection Agency (É.-U.), l'Organisation mondiale de la santé et d'autres organismes. On n'a pas voulu commenter tous les principaux documents consultés. Par contre, des travaux ou études originaux considérés comme étant les plus importants y sont inclus, ainsi que tout autre ouvrage important qui a été publié après ces documents relatifs aux critères.

2 Normes de qualité de l'air

Les normes ou les objectifs de qualité de l'air ambiant peuvent varier selon les pays et dans un même pays, au fur et à mesure que de nouvelles connaissances sont acquises. Leur but à long terme est de protéger la santé humaine contre les répercussions nuisibles. On a également considéré les répercussions nuisibles des polluants sur la végétation, la propriété matérielle, la visibilité et sur l'environnement en général. Le concept d'une norme visant à protéger les récepteurs d'une nuisance significative repose sur des données statistiques qui représentent un ensemble de cas singuliers de nuisance significative. L'adoption d'une norme ne garantie pas la protection de tous les individus. Les paragraphes suivants constituent une brève synopsis des normes de la qualité de l'air applicables au monoxyde de carbone, au dioxyde d'azote, aux hydrocarbures et aux oxydants photochimiques. Aucune norme de qualité de l'air n'a été imposée au Canada pour limiter les émissions des particules provenant des systèmes d'échappement de moteurs diesel ou concernant les dépôts acides.

Monoxyde de carbone. Les concentrations dites admissibles de carboxyhémoglobine (COHb) dans le sang ont diminué au cours de la dernière décennie, ce qui a contribué à abaisser les valeurs de référence existantes et à diminuer la rigueur des normes proposées. On est également de plus en plus conscient de l'accumulation rapide de COHb chez les personnes physiquement actives. Actuellement la limite maximale admissible est de 34 mg/m^3 (30 ppm) de CO pendant une heure. Cette limite recommandée en 1971, excède de 6 mg/m^3 (5 ppm) les plus récentes valeurs recommandées par l'Organisation mondiale de la santé (OMS) et les normes américaines proposées. Il en va de même pour la limite canadienne admissible dans le cas d'une exposition de huit heures, qui est plus forte que la norme américaine existante et la recommandation faite par l'OMS en 1979. Seule la norme japonaise pour une exposition de huit heures, publiée en 1971, est plus forte que la norme homologue canadienne.

Toutes les normes pour le CO, imposées ou recommandées, visent à protéger contre les répercussions nuisibles les personnes vulnérables, comme celles qui souffrent d'affections cardiaques et d'anémie. La méthode américaine est fondée sur le concept d'une marge de sécurité déterminée pour les groupes vulnérables qui visitent certains emplacements en haute altitude (par exemple, un cardiaque de New York en visite à Denver) et pour les foetus. Le modèle de relations entre l'exposition au CO et les concentrations sanguines de COHb comporte un certain degré d'incertitude. L'un des aspects étudiés actuellement par l'EPA et le Sous-comité canadien des objectifs nationaux de qualité de l'air est la sensibilité des concentrations du COHb prévues à l'aide du modèle Coburn lorsqu'elles sont associées à des concentrations variables plutôt qu'à des concentrations statiques de CO et à des paramètres physiologiques différents.

Dioxyde d'azote. Dans tous les pays, les normes de l'exposition à long terme au NO_2 voisines de 100 mg/m^3 (0,05 ppm) ont pour but de protéger la santé de certains groupes vulnérables de la population. Dans les pays qui ont publié des normes pour l'exposition à court terme (moyennes journalière ou d'une heure), les critères fondamentaux portent sur les répercussions sur la santé. On a étudié en outre, le rôle joué par le NO_2 dans l'évolution de la pollution photochimique (Japon et République fédérale allemande), les dommages à la végétation et la perception sensorielle (Canada). Les critères scientifiques disponibles pour les normes d'exposition à long terme basées sur les répercussions sur la santé sont très ténus et, à partir de 1977, l'Organisation mondiale de la santé a constaté que l'information n'était pas suffisante pour recommander des valeurs de référence pour la qualité de l'air. L'étude des critères scientifiques faite par l'EPA est également parvenue à cette conclusion mais, comme une norme annuelle était appliquée, on pensait qu'elle pouvait servir provisoirement de protection contre les concentrations maximales à court terme de NO_2. Un facteur de sécurité appréciable compris entre 3 et 5 a été incorporé dans la ligne directrice de l'OMS relative à la forte activité biologique en une heure du NO_2.

Hydrocarbures. Il n'existe pas d'objectifs de qualité de l'air pour les hydrocarbures au Canada. En 1983, les États-Unis ont annulé leur norme relative aux hydrocarbures autres que le méthane, qui était de 160 $\mu g/m^3$ (0,24 ppm) en moyenne pour trois heures (de 6 h 00 à 9 h 00). Cette norme comportait une caractéristique spéciale : les concentrations n'étaient pas basées sur les répercussions directes des hydrocarbures sur la santé ou le bien-être, mais avaient pour but de fournir un moyen de se conformer à la norme pour l'ozone.

Oxydants photochimiques. La chronologie des événements qui ont conduit à l'établissement des normes et à la recommandation de lignes directrices indique des tendances conflictuelles. En 1979, les États-Unis ont abaissé leur norme portant sur une heure de 160 $\mu g/m^3$ à 240 $\mu g/m^3$ (de 80 à 120 pp 10^9) tandis que, la même année, l'OMS abaissait ses lignes directrices pour la protection de la santé humaine de la plage des 200 - 250 $\mu g/m^3$ (100 - 125 pp 10^9) à celle des 100 - 200 $\mu g/m^3$ (50 - 100 pp 10^9). Au Canada, l'objectif maximal admissible est de 160 $\mu g/m^3$ (80 pp 10^9) pour une concentration moyenne d'une heure. Ces interprétations différentes sont essentiellement basées sur le même ensemble de critères scientifiques quoique l'OMS tende à utiliser plus que les États-Unis des données animales à l'appui de ses recommandations.

En général, les normes pour l'ozone ont été établies principalement pour protéger la santé publique. Au Canada, les dommages causés à la végétation ont également été pris en considération lors de l'établissement de concentrations maximales admissibles. En Ontario, les critères de qualité de l'air pour l'ozone visent principalement à éviter les dommages à la végétation.

3 Répercussions nuisibles

Les critères de qualité de l'air sont les répercussions ou réponses observées d'une ou plusieurs substances atmosphériques sur un récepteur ou une population définis dans des conditions déterminées. Ces critères servent de base à l'établissement des normes et, dans les cas où l'information est complète, correspondent aux relations "dose-réponse". Les paragraphes suivants constituent un résumé des répercussions nuisibles des polluants atmosphériques émis par les automobiles sur des récepteurs ou des populations.

3.1 **Santé.** Le monoxyde de carbone (CO) a des répercussions délétères sur la fonction cardiaque et est des plus dangereux pour les personnes atteintes d'une maladie cardiovasculaire. Le monoxyde de carbone a également des répercussions nuisibles sur le rendement au travail et les fonctions du système nerveux central. Une diminution de la vigilance ou du rendement lors de l'accomplissement de tâches complexes peut s'avérer importante au niveau de la population en général lorsque celle-ci est exposée à de faibles concentrations de CO, et ses effets possibles peuvent se faire particulièrement sentir sur l'aptitude à conduire des véhicules automobiles. Toutefois, aucune répercussion de ce type n'a été clairement documentée. Les femmes enceintes, les foetus et les nouveaux-nés sont très sensibles à la toxicité du monoxyde de carbone mais la quantification précise d'un risque accru n'est pas encore possible pour ce groupe.

L'exposition au dioxyde d'azote (NO_2) affecte la perception sensorielle et cause l'irritation des muqueuses du tractus respiratoire. Les répercussions nuisibles du NO_2 sur la fonction pulmonaire peuvent avoir une importance particulière pour les asthmatiques. Il semble également que le NO_2 augmente la susceptibilité à l'infection et il est possible qu'il soit responsable de l'augmentation de l'incidence de la maladie respiratoire, plus particulièrement chez les enfants.

L'exposition à l'ozone peut provoquer des anomalies dans différents organes. Le système respiratoire constitue, encore une fois, le site des plus grands dommages. L'ozone nuit à la fonction pulmonaire, l'importance de ses répercussions dépend de différents facteurs et notamment de la susceptibilité individuelle et du niveau d'activité physique. La diminution du rendement, l'exacerbation de l'asthme et l'augmentation de la susceptibilité à l'infection ont également été reliées à l'exposition à l'ozone.

On sait que les gaz d'échappement des moteurs diesel contiennent une forte concentration de particules qui ont été reliées à l'augmentation de la mortalité chez les personnes âgées et les malades atteints de maladies respiratoires (bronchite chronique, emphysème, etc.). On sait également que l'exposition aux particules cause l'exacerbation de la maladie chez ces malades et des études ont relié la fréquence des attaques d'asthme et l'incidence des maladies respiratoires aiguës chez les enfants à l'exposition aux particules. Par contre, on ne sait pas encore dans quelle mesure les fines particules, qui composent la majeure partie des émissions des moteurs diesel, peuvent influer sur les maladies respiratoires. Toutefois, la principale préoccupation relative à la santé est le potentiel cancérigène des émissions des moteurs diesel. Actuellement, aucun signe convaincant n'indique clairement l'existence de ce danger, qui ne peut, à plus forte raison, être quantifié.

Aux concentrations ambiantes, les hydrocarbures considérés comme une catégorie ont peu de répercussions directes sur la santé. Toutefois, certains d'entre eux peuvent être dangereux à des concentrations voisines de celles de l'air ambiant. Le benzène, par exemple, qu'on sait capable de causer de sérieux désordres sanguins, notamment la leucémie, peut inquiéter dans certaines situations particulières. Les aldéhydes, le formaldéhyde par exemple, qui peuvent être formés à partir des hydrocarbures émis dans les gaz d'échappement des véhicules automobiles, peuvent irriter les muqueuses ou causer des attaques d'asthme chez les personnes sensibles. La quantification de ces effets n'est pas possible actuellement.

Les précipitations acides peuvent également affecter indirectement la santé en augmentant le danger d'empoisonnement par le mercure et le plomb ou le danger d'intoxication par d'autres éléments dans certaines régions et chez certaines populations.

3.2 **Systèmes terrestres.** Les études des répercussions des polluants atmosphériques sur les systèmes terrestres ont porté principalement sur la végétation. La blessure visible de la feuille exposée au NO_2 est le symptôme le plus facilement décelable et le plus fréquemment signalé et a fait l'objet de nombreux rapports sur les dommages causés aux cultures commerciales. Certaines études ont toutefois montré que des diminutions de la croissance et du rendement des plantes peuvent se produire sans qu'apparaissent ces symptômes visibles.

L'exposition à court terme à des maximums de NO_2 peut causer autant de dommages, sinon plus, à la végétation que l'exposition continuelle à ce gaz au cours d'une saison de croissance. Les diminutions de la croissance et du rendement peuvent se reproduire en n'étant accompagnées que de blessures foliaires minimes ou en l'absence de ces blessures; il est également possible de déceler des blessures foliaires graves sans observer de répercussions significatives sur les rendements agricoles.

Certaines études des expositions combinées au NO_2 et au SO_2 ont révélé que le seuil de blessure du NO_2 peut être diminué significativement en présence de SO_2. Les réponses à ces mélanges sont reliées aux rapports des concentrations, à la séquence des expositions et à d'autres variables. Dans certaines régions d'Amérique du Nord, les concentrations ambiantes sont comprises dans des plages de concentrations auxquelles des blessures observables ont été causées.

Certaines données disponibles semblent indiquer que l'ozone (O_3) est le plus pénétrant et le plus dommageable, sur le plan économique, des oxydants photochimiques. Les répercussions de l'ozone sur les plantes se manifestent notamment par des altérations métaboliques, la diminution de la production primaire et la modification subséquente du compartimentage des produits de la photosynthèse et des diminutions connexes de la biomasse et(ou) du rendement. La qualité de la plante subit d'autres répercussions, comme la blessure foliaire ou l'altération de ses éléments nutritifs. D'après certaines études la blessure foliaire constitue un indicateur utile de la présence d'oxydants photochimiques, mais comme cette blessure peut être également causée par d'autres stress on ne peut pas toujours se fier à ce facteur pour prévoir les répercussions des oxydants sur la croissance, le rendement ou la qualité. Certains signes semblent indiquer actuellement que l'ozone que l'on trouve dans l'air ambiant de nombreuses régions d'Amérique du Nord est suffisant pour diminuer la croissance et le rendement de la végétation et que certaines cultures sensibles subissent des modifications significatives dues à l'exposition saisonnière pendant les sept heures journalières médianes à des concentrations d'O_3, comprises entre 80 et 200 $\mu g/m^3$ (0,04 et 0,10 ppm).

Certains signes semblent indiquer que de grandes parties des écosystèmes forestiers de la zone tempérée subissent des perturbations reliées aux oxydants qui apportent des modifications significatives à la forêt. Certaines diminutions de la production et de la diversité sont évidentes, les forêts primaires et celles qui leur ont succédé en partie étant particulièrement en danger. On pense que les stress continuels à long terme diminuent la couverture foliaire totale et la richesse des espèces et augmentent la dominance des espèces tolérantes aux oxydants. Au fil du temps, certaines modifications progressives et subtiles du métabolisme, de la croissance et de la composition des forêts dans de vastes régions de la zone tempérée sont considérées par les chercheurs comme étant beaucoup plus importantes que la destruction sur de courtes périodes, plus dramatique, des forêts au voisinage immédiat des sources ponctuelles d'oxydants.

Les composés nitriques des précipitations acides ne sont probablement pas significatifs comme source d'éléments nutritifs pour les cultures agricoles mais peuvent fournir jusqu'à 40 p. 100 des besoins en azote des forêts. Il existe actuellement peu de signes certains prouvant que les concentrations existantes d'azote dans les précipitations acides ont des répercussions délétères sur la végétation terrestre, mais ces concentrations peuvent modifier la productivité de la forêt et des plantes agricoles par impaction directe, par des modifications de la structure et des processus du sol ou par lessivage des minéraux des bassins versants. La blessure foliaire n'a pas été documentée sur place à cause des dépôts acides, mais certains traitements appliqués dans des conditions contrôlées, comportant des précipitations acides simulées, ont causé, dans certains cas, des blessures foliaires et des diminutions ou stimulations de la croissance. Les études terminées sont trop peu nombreuses pour pouvoir évaluer avec précision les répercussions terrestres des précipitations acides.

3.3 **Systèmes aquatiques.** Dans l'est de l'Amérique du Nord, les apports sulfurés acides excèdent annuellement les apports azotés acides dans les précipitations acides et le produit annuel net de ces anions dans les cours d'eau et les lacs est constitué surtout de sulfate. Certains chercheurs ont conclu que puisque le nitrate atteint les eaux superficielles en petites quantités par rapport à la charge annuelle de ces eaux et ne s'y accumule pas, son influence sur l'acidification à long terme des eaux superficielles est plus petite que celle du sulfate.

L'acide nitrique peut jouer un rôle plus significatif dans l'acidification à court terme liée à la fonte printanière du manteau nival contenant du nitrate. C'est la raison pour laquelle le nitrate peut être épisodiquement important parce qu'il accentue la diminution existante du pH causée par le sulfate. À ce jour, certaines études de ce phénomène ont constaté que les concentrations de sulfate excèdent encore celles du nitrate mesurées sur une base équivalente, même pendant l'écoulement printanier.

Les conclusions des documents de référence publiés permettent d'affirmer que la plus grande partie de l'augmentation de l'acidité observée dans les réseaux d'eaux superficielles était due aux modifications de la concentration de sulfate attribuées aux dépôts du sulfate et de l'acide sulfurique. Les acides sulfurique et nitrique contribuent à l'acidification des eaux superficielles pendant des périodes de diminutions du pH et de stress pour le poisson. Il n'existe actuellement pas de signe certain permettant de prévoir une diminution appréciable de l'acidification à long terme des lacs ou des cours d'eau à cause d'une diminution des apports de nitrate.

L'azote atmosphérique peut constituer une part importante de la charge des Grands lacs et des eaux canadiennes. Dans la plupart des lacs, l'eutrophisation est limitée dans une grande mesure par le phosphore présent, il est donc invraisemblable que l'azote atmosphérique soit un élément majeur dans le processus d'enrichissement des lacs. Il faut considérer comme des exceptions les écosystèmes des côtes, des estuaires et des lacs eutrophisés qui peuvent connaître une stimulation de la production aquatique lorsque les apports de nitrate augmentent.

3.4 **Substances artificielles.** Les dommages causés aux substances artificielles par l'ozone et le dioxyde d'azote ont été documentés pour certaines concentrations observées dans l'atmosphère ambiante. Toutefois, certaines précautions doivent encore être prises pour appliquer à des expositions réelles la documentation sur les dommages matériels élaborée dans des études de laboratoire.

Les fibres cellulosiques (coton et rayonne à la viscose) teintes à l'aide de colorants directs, colorants à cuve et colorants réagissant sur la fibre, subissent une grave décoloration à l'exposition en cuve à 940 $\mu g/m^3$ (0,5 ppm) de NO_2 en atmosphère très humide (90 p. 100) et très chaude (30 °C). Une décoloration significative est observée après une exposition de 12 semaines à 100 $\mu g/m^3$ (0,05 ppm) NO_2 dans les mêmes conditions d'humidité et de température élevées. Sur le nylon, les colorants acides se décolorent lorsqu'ils sont exposés à une concentration de 200 $\mu g/m^3$ (0,1 ppm) de NO_2 dans les mêmes conditions environnementales. Le jaunissement des tissus blancs en fibres de polyuréthane segmentées, coton caoutchouté, acétate blanchi optiquement et nylon a été documenté par des études en laboratoire où ils ont été exposés pendant 8 heures à une concentration de 400 $\mu g/m^3$ (0,2 ppm) de NO_2. Certains nitrates ont été rendus responsables de la rupture de fils en alliage de nickel et de laiton utilisés dans l'équipement téléphonique, mais il n'existe pas d'autres rapports portant sur la corrosion des métaux.

Les nitrates étant plus hygroscopiques que les chlorures ou les sulfates, l'utilisation des premiers peut abaisser le seuil de la corrosion des métaux par l'humidité.

Les seules répercussions quantifiées de l'ozone sur les substances artificielles sont celles des produits utilisés pour fabriquer les pneus. Le caoutchouc naturel se fissure en présence de concentrations d'ozone aussi faibles que 40 $\mu g/m^3$ (20 pp 10^9). Les pneus sont protégés de la dégradation grâce à l'addition d'agents antiozone qui inhibent les réactions du caoutchouc avec l'ozone. L'ozone a des répercussions sur les colorants textiles qui sont altérés dans des conditions spéciales qui ne peuvent pas être facilement généralisées dans l'environnement urbain. Toutes ces répercussions ont été observées à de hautes températures et dans des conditions d'humidité relativement élevées. Des travaux récents ont indiqué qu'il peut exister une association significative entre l'érosion de la peinture au latex et l'humidité relative en présence d'ozone, mais des études plus approfondies sont nécessaires pour établir définitivement une fonction "dose-réponse" pour le dommage.

3.5 **Visibilité.** La visibilité constitue un élément important dans l'appréciation de la qualité de l'air par la population en général et les organismes gouvernementaux. Au Canada, la protection contre les obstructions de la visibilité est l'un des critères sur lesquels est fondé l'objectif de qualité maximale de l'air relativement aux particules en suspension. Récemment, l'Environmental Protection Agency et le Conseil national de recherches du Canada ont recommandé que des normes (objectifs) soient élaborées pour les particules fines, la visibilité servant de critère, d'après une relation relativement bien définie entre la portée visuelle et la concentration massique des particules fines. Dans de nombreux cas, ces particules fines sont des produits photochimiques des émissions gazeuses. La méthode d'estimation de la portée visuelle à partir de la concentration massique de particules fines est assez bien connue. Une relation semi-empirique peut être utilisée comme indicateur de l'importance relative des différentes espèces chimiques présentes sous forme de particules fines en tant qu'obstruction de la visibilité. Dans l'est de l'Amérique du Nord, l'importance de l'action du sulfate sur la diminution de la visibilité a été confirmée tandis que celle des nitrates, des composés organiques et du carbone (suie) dépend plus de l'emplacement.

Le Protocole qui a fait l'objet d'une entente récente entre le Canada et les États-Unis relativement au transport sur de longues distances des polluants a permis de conclure que "les données disponibles semblent indiquer que les nitrates présents surtout à l'état de vapeur n'ont, pour la plupart, que peu de conséquences sur la visibilité dans l'est de l'Amérique du Nord".

L'effet du dioxyde d'azote sur la diminution de la portée visuelle est petit mais, ce composé peut, dans certaines circonstances, provoquer l'apparition d'une brume sèche brunâte; dans la majorité des cas, toutefois, cette brume sèche est attribuable à la diffusion de la lumière solaire par les particules.

3.6 **Répercussions indirectes.** La formation d'un produit de réaction secondaire qui a lui-même des effets directs sur la santé humaine dans les cinq domaines examinés sommairement dans les paragraphes précédents comportent des répercussions indirectes. Celles-ci sont notamment la formation d'oxydants photochimiques à partir des émissions primaires d'hydrocarbures et d'oxydes d'azote en présence de lumière solaire et la formation de nitrates et d'acide nitrique à partir des émissions d'oxydes d'azote. Les produits finals ont tous des répercussions nuisibles connues ou soupçonnées sur les différents récepteurs.

La relation quantitative entre les précurseurs (NO_x et hydrocarbures) et les produits finals (O_3, HNO_3) est complexe et n'est pas complètement comprise.

Le rôle des émissions de NO_x dans la formation des oxydants photochimiques prête à controverse. Certaines études semblent indiquer que la diminution des concentrations de NO_x présentes favorise la formation de l'ozone.

Tous les processus de conversion dans le phénomène des précipitations acides exigent des substances oxydantes comme le peroxyde d'hydrogène, l'ozone et le radical hydroxyle. La production de ces composés est directement reliée à la catégorie des hydrocarbures réactifs polluants. Les conversions des polluants atmosphériques en aérosols acides se produisent à des vitesses différentes qui dépendent des quantités relatives et absolues de dioxyde de soufre, d'oxydes d'azote, d'hydrocarbures et de particules fines présentes dans les émissions. Les conditions météorologiques influencent également fortement les conversions générales. Le processus de conversion de l'acide au cours d'un événement (période courte) peut être caractérisé par la concurrence du SO_2 et des NO_x pour fixer les oxydants et l'inhibition de la formation d'un acide par un autre composé présent. Toutefois, sur une période longue et à l'échelle d'une grande région, il est possible de considérer ces réactions comme étant linéaires. En d'autres mots, la concentration d'acide formé peut être directement proportionnelle à la quantité de son précurseur émise.

2. AIR POLLUTION

2.1 Concepts of Air Pollution

Air is defined as the relatively constant mixture of gases enveloping the earth up to about 8 kilometres above the surface. The components of clean or unpolluted air, given in Table 2.1-1 consist mainly of oxygen (21 percent by volume of air), nitrogen (78 percent) and trace gases (1 percent). Oxygen is very reactive and chemical reaction with this substance (oxidation) is ever present involving both living and inert matter. Nitrogen, which is relatively inert, only enters chemical reactions in special circumstances such as in presence of lightning or at high temperatures. The major trace variable of air is water vapour which ranges in concentration from about 200 to 300 ppm (0.02 to 0.03%) in dry regions to 50,000 or 60,000 ppm (5 to 6%) in a warm humid climate. Water vapour plays an important role as solvent and catalyst for many thermal and photochemical reactions in the atmosphere. The gases, carbon dioxide and methane, are of potential importance in maintaining climatic stability while the noble gases-argon, helium, neon, krypton, xenon-are inert and for all practical purposes can be ignored in atmospheric processes.

In addition to the above-mentioned substances, numerous other gases may be delineated such as sulphur dioxide, carbon monoxide and ozone, along with particles present in clean air at very low concentrations. The atmospheric background level of these compounds varies over water and land and with latitude. This originates from natural sources and chemical processes in air. There is never truly clean air insofar as decaying vegetable matter, decaying dead animals, forest fires, and volcanoes emit gaseous and particulate compounds which may exist temporarily in local areas at high levels.

TABLE 2.1-1

THE GASEOUS COMPOSITION OF UNPOLLUTED AIR

The Gaseous Composition of Unpolluted Air (Dry Basis).*

	ppm(vol.)	% by volume	µg/m^3	% by weight
Nitrogen	780,900	78.090	8.95×10^8	75.540
Oxygen	209,400	20.940	2.74×10^8	23.126
Water	-	-	--	
Argon	9,300	0.930	1.52×10^7	1.283
Carbon dioxide	315	0.032	5.67×10^5	0.048
Neon	18	0.002	1.49×10^4	0.001
Helium	5.2	<0.001	8.50×10^2	<0.001
Methane	1.0-1.2	<0.001	$6.56\text{-}7.87 \times 10^2$	<0.001
Krypton	1.0	<0.001	3.43×10^3	<0.001
Nitrous oxide	0.5	<0.001	9.00×10^2	<0.001
Hydrogen	0.5	<0.001	4.13×10^1	<0.001
Xenon	0.08	<0.001	4.29×10^2	<0.001
Organic Vapours	ca. 0.02	<0.001	--	

The Gaseous Composition of Unpolluted Air (Wet Basis).*

	ppm(vol.)	% by volume	µg/m^3	% by weight
Nitrogen	756,500	75.650	8.67×10^8	74.085
Oxygen	202,900	20.290	2.65×10^8	22.644
Water	31,200	3.120	2.30×10^7	1.965
Argon	9,000	0.900	1.47×10^7	1.256
Carbon dioxide	305	0.031	5.49×10^5	0.047
Neon	17.4	0.002	1.44×10^4	0.001
Helium	5.0	0.001	8.25×10^2	<0.001
Methane	0.97-1.16	<0.001	$5.35\text{-}7.63 \times 10^2$	<0.001
Krypton	0.97	<0.001	3.32×10^3	<0.001
Nitrous oxide	0.49	<0.001	8.73×10^2	<0.001
Hydrogen	0.49	<0.001	4.00×10^1	<0.001
Xenon	0.08	<0.001	4.17×10^2	<0.001
Organic Vapours	ca. 0.02	<0.001	--	

* from "Fundamentals of Air Pollution". A.C. Stern, H.C. Wohlers, R.W. Boubel and W.P. Lowry, Academic Press (1973) and "Chemical Compounds in the Atmosphere" T.E. Graedel, Academic Press (1978).

Air pollution may be considered as the presence in the outdoor atmosphere of a substance (gaseous or particulate) or mixture of substances, added in sufficient amounts to produce a measurable effect on man, animals, vegetation, or materials. What is considered as a pollutant depends upon recognition of the substance (or substances) which cause adverse effects. Air quality refers to the characteristics of ambient air that are required to ensure against adverse effects. Thus air quality is poor if the concentration of a pollutant (or pollutants) in air exceed the level at which it is considered to have an adverse effect.

There are two basic categories of air pollutants: primary and secondary. Primary pollutants such as carbon monoxide and hydrocarbons are directly released into the air from fuel combustion in stationary and mobile sources or from other processes. However, under the proper conditions, some of these primary pollutants can undergo chemical reactions and transformations in the atmosphere to produce secondary pollutants such as ozone and nitric acid. Air pollutants can result from both natural and man-made sources and in some cases, there is no clear distinction between the natural and man-made air pollutants. The atmosphere serves as a delivery system from emission sources to the receptor. Once the substance is in the atmosphere, what happens to it depends on its physical and chemical characteristics rather than whether it was of natural or man-made origin. Man's activities that lead to air pollution are usually a result of overloading the atmosphere's capability to dilute, to transform emissions to innocuous compounds, or to recycle substances. The associated elevation of pollutant levels above that in clean air may cause adverse effects.

The final portion of the atmospheric cycle of a substance consists of several removal processes. These various processes serve to cleanse the atmosphere of accumulated material and deliver it to the surface of the earth. Removal processes may be classified into three types: wet removal, dry deposition, and chemical transformations.

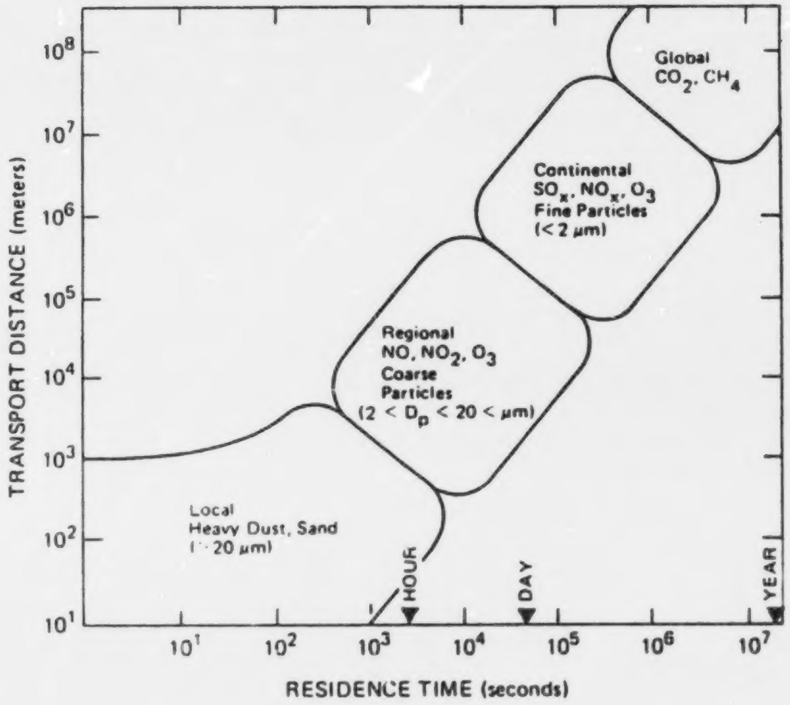
Wet removal is associated only with precipitation, whereas dry removal processes go on all the time. Chemical transformations in the atmosphere are thought of as a removal process since they transform one species into another. The hydroxyl radical (OH) reactions appear to be the dominant gas-phase process by which most trace gases (i.e. carbon monoxide, hydrocarbons, nitrogen dioxide, and sulphur dioxide) are consumed in the atmosphere (Niki *et al.*, 1972; Demerjian *et al.*, 1974; Calvert *et al.*, 1978).

One of the more important questions to be answered when a pollutant has been released into the atmosphere is what area will be affected. To attempt an answer consideration must be given to the strength of the source, the way in which an effect is made manifest, whether the effects depends on airborne concentration or deposition, whether atmospheric processes are involved and meteorological considerations. Air pollution is in the first instance a local problem; however, the pollutants gradually mix into larger and larger volumes of air until they are distributed over very large areas. This leads to consideration of the properties of air pollutants on progressively larger spatial scales.

Air pollution problems may be grouped into four spatial scales: local (0 to 50 km), regional (50 to 500 km), continental (500 to 5000 km) and global (greater than 5000 km). One of the aspects that determines the zone of influence of an air pollutant is atmospheric residence time. An obvious requirement for a large-scale pollution problem is that a substance must have an atmospheric life time sufficiently long to be transported over long distances. Figure 2.1-1 gives examples of atmospheric substances with various residence times and the corresponding air pollution scale. A compound with a residence time of years, such as carbon dioxide, has time to mix throughout the atmosphere and become globally distributed. For compounds with shorter residence times their potential zones of influence are reduced accordingly. For example, ozone can be expected to have a potential effect up to the continental scale while dust raised from streets is only noticed up to several tens of metres. At continental

FIGURE 2.1-1

DISPERSION OF POLLUTANTS INTRODUCED INTO THE ATMOSPHERE AS DETERMINED BY RESIDENCE TIME *



Note: D_p refers to particle diameter

*Reproduced from NRC, 1981

scales, especially at mid-latitudes of the Northern Hemisphere, removal of some substances by precipitation becomes important.

On a local scale pollutant levels can readily be distinguished from global, continental, and regional levels. For example, congestion of automobiles at intersections in downtown cores, gives rise to levels of pollutants that are easily distinguishable from average levels over the city and the rural countryside.

Associated with the four scales of air pollution is the time for effects to be noticed at each level. On the local scale, deleterious effects could arise within hours while at the opposite end of the spectrum, global effects may not be noticed even after decades. High levels of some air pollutants in urban areas (the local scale) have been associated with health effects. This has resulted in the formulation of air quality standards in various countries. However, even at this scale generally accepted quantitative assessment of the range of effects are still not available. The next section addresses the air pollutants associated with automobiles and their potential scale of influence.

2.2 Automotive Air Pollutants

2.2.1 Automotive Emissions

The gasoline powered automobile is a source of three primary air pollutants-carbon monoxide, nitrogen oxides, and hydrocarbons. The hydrocarbon mixture generated by motor vehicles is complex with over 300 identified compounds (Hampton et al., 1982). It is well known that ozone and other photochemical oxidants are produced by the action of sunlight on mixtures of hydrocarbons and nitrogen oxides (Haagen-Smit and Wayne, 1976; Haagen-Smit, 1952). The automobile's importance as a contributor to the formation of photochemical oxidants relative to other sources varies according to the characteristics of an area. Automobile emissions come from more than the tailpipe; hydrocarbons are also released through evaporation from the fuel tank and carburetor.

Exhaust emissions from a diesel-powered automobile are similar to those from its gas-powered counterpart with one major exception. Diesel engines release substantial amounts of particles (soot) which contain organic compounds.

The 1976 Canadian Emission Inventory (EPS, 1981a) of anthropogenic sources states: "Nitrogen oxide emissions are mainly from gasoline-powered motor vehicles (26.8% of total) and industrial combustion (19.7% of total). Gasoline-powered motor vehicles and forest fires are important sources of hydrocarbon emissions (33.1% and 31.3% of total). These two sources are also major contributors of CO emissions (46.6% and 31.4% of total)."

2.2.2 Carbon Monoxide

Carbon monoxide (CO) is a colourless, odourless, and tasteless gas. After carbon dioxide (CO_2), CO is the pollutant which is emitted into the air in the largest quantities. Man-made emissions are not the only sources; oxidation of methane in the atmosphere, oceans, and degradation of chlorophyll are natural sources. Overall, the man-made source of CO may be compared in magnitude with natural sources (Freyer, 1979). The mean lifetime of carbon monoxide in the atmosphere is around 60 days and its concentration in unpolluted air of the mid-latitudes of the Northern Hemisphere is around 0.23 to 0.29 mg/m^3 (0.20 to 0.25 ppm) (Ehhalt, 1981).

For many years, it was believed that CO did not participate in photochemical reactions. However, experiments in the early 1970's demonstrated that its presence could speed up the oxidation of nitric oxide (NO) to nitrogen dioxide (NO_2), thus hastening the appearance of ozone (Westberg et al., 1971). This reaction is sufficiently fast to be important in the urban atmosphere with a CO concentration of 6 mg/m^3 (5 ppm) (National Academy of Sciences, 1977).

If no hydrocarbons were present in the atmosphere but carbon monoxide and nitrogen oxides were present, significant ozone concentrations would develop. With hydrocarbons present, the addition of CO does not have a strong effect on oxidant concentrations unless it is present in levels of about 2290 mg/m^3 (2000 ppm) (ibid, 1977) which is much higher than actual monitored values in urban areas. Fishman and Crutzen (1978) speculate that oxidation of CO by the hydroxyl (OH) radical in the presence of NO_x may lead to significant ozone production in the global atmosphere.

Concentrations of CO near man-made sources vary with time, season, and geographic location. The dispersion and dilution of CO until it reaches background levels is affected by wind, atmospheric stability, vertical mixing height and ambient temperature. The following automobile related scenarios may result in unusually high ambient levels of CO (EPA, 1979): (1) on a city freeway where traffic is stopped, CO levels may exceed 50 mg/m^3 (44 ppm); (2) inside a closed automobile where cigarettes are smoked, CO may exceed 100 mg/m^3 (87 ppm); and (3) in heavily travelled vehicular tunnels, a one-hour maximum of 250 mg/m^3 (218 ppm) CO was recorded. The highest levels of carbon monoxide measured in Ontario in 1980 were at 381 Yonge Street in Toronto. This station, located close to a major traffic artery and within an urban street canyon, measured an annual average of 4.8 mg/m^3 (4.2 ppm), a maximum 8-hour average of 29.9 mg/m^3 (26 ppm), and a maximum 1-hour average of 43.7 mg/m^3 (38 ppm) (MOE, 1981). No other station exceeded these 8-hour and 1-hour values. The arithmetic mean of monitors in rural locations was 0.12 mg/m^3 (0.1 ppm) which may be considered the background level.

2.2.3 Nitrogen Oxides

There are numerous oxides of nitrogen (NO_x) but for all practical purposes, NO_x is taken to be the sum of nitric oxide (NO) and nitrogen dioxide (NO_2). Nitric oxide is a colourless, odourless gas which is only slightly soluble in water. In most urban atmospheres, NO is readily converted (oxidized) to NO_2. Nitrogen dioxide has a reddish-brown colour and a pungent odour. Although the boiling point of NO_2 is 21°C, its low partial pressure prevents condensation in the atmosphere. NO_2 is involved in photochemical reactions because of its strong absorption of sunlight which leads to subsequent decomposition to NO and atomic oxygen (O). Several other compounds derived from NO_x are of importance in air pollution. These substances include nitrates (NO_3), nitric acid (HNO_3), N-nitroso compounds, and organic compounds such as peroxyacetylnitrates (PAN). These compounds are discussed in the sections on photochemical oxidants and acid precipitation.

Natural or clean air levels of nitrogen oxides result from forest fires, lightning, microbial activity in soils and possibly oxidation of ammonia in the atmosphere. Estimates of natural emissions of NO_x in Canada range from about 1 to 2.7 million tonnes per annum (Logan , 1982; EPS 1981b); however, there are large uncertainties associated with these values. The 1976 inventory of Canadian anthropogenic sources estimated annual NO_x emissions to be about 1.9 million

tonnes with gasoline-powered automobiles contributing approximately 1/2 million tonnes (EPS, 1981a). Thus the gasoline-powered automobiles release 12 to 20 percent of the total NO_x emissions (natural and man-made) in Canada. A unique difference between natural and man-made sources is that the former are widely distributed throughout the country while the latter are usually concentrated in urban and industrial centres. This concentration of anthropogenic sources leads to higher concentrations of NO_x than are found in clean background air. The concentrations of NO and NO_2 in clean air over land are considered to be less than 0.001 ppm (Logan, 1982).

Nitrogen oxides are released to the atmosphere primarily in the form of NO. Only about 5 to 10 percent by volume of the total emissions of NO_x from combustion sources is in the form NO_2 (EPA, 1982). Nitric oxide reacts rapidly with ozone (O_3) to form NO_2 by:

$$NO + O_3 \longrightarrow NO_2 + O_2 \qquad (1)$$

which in turn is photodissociated into nitric oxide and atomic oxygen:

$$NO_2 + h_\nu \longrightarrow NO + O \qquad (2)$$

Oxygen atoms in air react predominantly with molecular oxygen to regenerate ozone:

$$O + O_2 + M \longrightarrow O_3 + M \qquad (3)$$

where M represents a third compound (nitrogen, oxygen, water, etc.). The combination of the results of reactions 1, 2, and 3 causes generation of a small concentration of ozone directly related to the ratio of NO_2 to NO concentrations and the intensity of sunlight absorbed by NO_2 (reaction 2).

However, in a sunlight-irradiated, polluted atmosphere, nitric oxide may be converted rapidly to NO_2 by reaction with perhydroxyl and organic peroxy radicals by:

$$NO + HO_2 \longrightarrow NO_2 + OH \qquad (4)$$

$$NO + RO_2 \longrightarrow NO_2 + RO \qquad (5)$$

where R represents methyl (CH_3), ethyl (C_2H_5) and higher alkyl groups. During the day NO and NO_2 are interconverted on a time scale of minutes by reactions 1, 2, 4 and 5. NO_x can lead to formation of ozone in the atmosphere by reactions 1 and 2, provided NO_x levels are about 30 ppt (Logan et al, 1981).

Nitrogen oxides are removed from the atmosphere by conversion to nitric acid via the reaction:

$$OH + NO_2 \; (M) \longrightarrow HNO_3 + (M) \qquad (6)$$

followed by rainout or surface deposition of HNO_3. Logan et al. (1981) estimate for the unpolluted atmosphere that NO_x is converted to HNO_3 within 1 to 2 days at mid-latitudes in summer and within about 10 days in winter. Lifetimes of 4 to 8 hours were found in plumes from the urban areas of Boston and Philadelphia during summer (Spicer, 1982, Spicer and Sverdrup, 1980). Spicer's measurements show that NO and NO_2 are converted into HNO_3, peroxyacetylnitrate (PAN), and particulate nitrate.

Nitric acid concentrations as high as several ppb have been observed in urban areas and the ratio of PAN:HNO_3 varied between 0.5 and 3 (Spicer, 1977). Concentrations reported for rural sites in the eastern United States are about 1 ppb for HNO_3 and a few hundred ppt for PAN (Spicer and Sverdrup, 1980; Holdren et al 1982). The concentration of NO_2 in polluted air in Canada, United States and Europe sometimes exceeds 188 to 376 $\mu g/m^3$ (0.1 to 0.2 ppm), (Platt and Perner, 1980; EPS, 1981c).

Typical conditions for high NO_2 concentrations are usually a high flux of sunlight in combination with stagnant air. However, high concentrations of NO_2 can also occur during winter. Lindqvist et al. (1982) indicate that high NO_2 concentrations occur in Northern Europe during relatively long periods of time characterized by an inversion, no wind, temperatures around or below 0°C and relatively low photochemical activity. NO_2 concentrations of 188 to 564 $\mu g/m^3$ (0.1 to 0.3 ppm) in Göteborg, Sweden can last for several hours each day during an inversion period.

2.2.4 Hydrocarbons

The class of compounds known as hydrocarbons is unique among the emissions of automobiles. All pollutants except for hydrocarbons are believed to have direct effects on health, vegetation, or materials. Hydrocarbons mainly cause adverse effects indirectly through their contribution to the formation of photochemical oxidants. However, one member of this class, benzene, can be present in ambient air at levels that represent a potential adverse health effect (Tilton et al. 1981).

Vehicle exhaust gas contains low molecular-weight hydrocarbons such as methane, ethane, ethylene, acetylene, propylene, and olefins which are present in gasoline. The emissions also contain partially oxygenated organic compounds besides hydrocarbons such as aldehydes, ketones, alcohol ethers, esters, acids and phenols (National Academy of Sciences, 1976). Aldehydes are generally believed to be the most important of these organic compounds. Some of the aldehydes (e.g. formaldehyde) may also be deleterious to health. In addition, polycyclic aromatic hydrocarbons (PAH), such as benzo(a)pyrene, are products of incomplete combustion. The PAH's are of concern because of their mutagenic and carcinogenic properties.

Methane (CH_4) is the most abundant gaseous hydrocarbon in the atmosphere resulting from the bacterial decomposition of organic matter in oxygen deficient environments such as swamps. By comparison anthropogenic sources are of minor importance. The major process for removal of CH_4 from the atmosphere appears to be oxidation by the OH radical which eventually leads to formation of CO.

It is well established that hydrocarbons much more reactive than methane are emitted in significant quantities in vehicle exhaust, during handling of solvents, and from the petrochemical industry in general (EAG, 1981a). Reactivity of a hydrocarbon or organic compound is generally taken to be a measure of the extent of effect which the presence of a particular hydrocarbon has in the formation of reaction products in sunlight in the presence of nitrogen oxides and air. Hydrocarbon reactivity can be measured in terms of the rate of ozone formation or maximum concentration of ozone produced. One scheme, based on maximum one hour ozone concentrations observed in laboratory smog chamber tests, is used in Table 2.2-1. This table illustrates both the types of hydrocarbon emissions from automobiles and their reactivity. It is normally recognized that the hydrocarbons, methane, ethane, acetylene, and benzene have small or negligible reactivity (Classes 00 and 01 in Table 2.2-1). Highly reactive organic compound emitted by automobiles include the aldehydes and olefins (Class 5).

Non-methane hydrocarbons are also emitted by natural processes in vegetation, microorganism decomposition in soils and water body sediments, and forest fires (EAG, 1981b). The role of reactive natural hydrocarbons in photochemical pollution is conflicting. Dimitriades (1981) concluded from measured concentrations in combination with reactivity data that natural hydrocarbons are judged to contribute negligibly to urban ozone, and that their contribution to rural ozone is less certain but probably not major.

2.2.5 Photochemical Oxidants

The mixture of products resulting from reactive hydrocarbon interference in the NO_2 photolytic cycle is commonly called photochemical smog. The photochemical oxidants present in smog comprise ozone (O_3), peroxyacetylnitrates (PAN), and other organic compounds including aldehydes and alkyl nitrates. This section deals with ozone which is the most abundant photochemical oxidant.

TABLE 2.2-1

EXAMPLE OF AUTOMOBILE ORGANIC SPECIES PROFILES AND THE REACTIVITY CLASSIFICATION OF THE COMPOUNDS

Evaporative Emissions
from Gasoline Powered Automobiles

CHEMICAL NAME	PERCENT WEIGHT	REACTIVITY CLASSIFICATION
ISOMERS OF HEXANE	11.62	03
ISOMERS OF HEPTANE	4.78	03
ISOMERS OF OCTANE	4.63	03
ISOMERS OF NONAME	0.45	03
ISOMERS OF DECANE	0.68	03
N-BUTANE	11.88	03
ISOMERS OF PENTANE	36.78	03
ISOMERS OF BUTENE	4.51	05
ISOMERS OF PENTENE	7.22	05
1-HEXENE	2.34	05
ISOMERS OF OCTENES	0.15	05
HEPTENES	0.44	05
ISOMERS OF XYLENE	0.83	04
ISOMERS OF BUTYLBENZENE	0.04	04
ISOMERS OF TRIMETHYLBENZENE	2.54	05
TOLUENE	9.12	04
ETHYLBENZENE	0.20	04
BENZENE	1.79	01
TOTAL	100.00	

Gasoline Fuelled Automobile Exhaust
(with no Exhaust Controls)

CHEMICAL NAME	PERCENT WEIGHT	REACTIVITY CLASSIFICATION
ISOMERS OF HEXANE	4.15	03
ISOMERS OF HEPTANE	5.21	03
ISOMERS OF OCTANE	8.29	03
ISOMERS OF NONANE	1.23	03
ISOMERS OF DECANE	0.32	03
N-BUTANE	3.10	03
ISOMERS OF PENTANE	4.92	03
ETHANE	0.68	01
ISOMERS OF BUTENE	4.34	05
ISOMERS OF PENTENE	2.06	05
ETHYLENE	11.60	05
PROPYLENE	8.50	05
1-HEXENE	0.78	05
HEPTENES	1.02	05
ISOMERS OF XYLENE	3.62	04
ISOMERS OF BUTYLBENZENE	0.07	04
ISOMERS OF TRIMETHYLBENZENE	2.06	05
TOLUENE	11.91	04
ETHYLBENZENE	0.74	04
BENZENE	3.90	01
FORMALDEHYDE	4.70	05
METHANE	7.60	00
ACETYLENE	9.20	01
TOTAL	100.00	

Source: EAG, 1981a

Ozone is present naturally in air arising from such sources as stratospheric injection into the lower atmosphere and photochemical reactions. In the global atmosphere, O_3 has a lifetime of several weeks. Ozone is not uniformly mixed in the atmosphere. Near the earth's surface, its concentration is in the order of 40 to 120 $\mu g/m^3$ (20 to 60 ppb). Ozone has a special role in determining the reactivity of common trace gases in air. When O_3 is photochemically decomposed, an OH radical is formed. This radical is the primary controller of oxidation of many trace gases in the atmosphere such as carbon monoxide and nitrogen oxides. Ozone occurs at relatively high concentrations in the stratosphere where it is produced by ultraviolet radiation from the sun. It also occurs in high concentrations in polluted urban atmospheres and downwind of cities, being formed by the interaction of sunlight with the ozone precursors, reactive hydrocarbons and nitrogen oxides.

Most of the reactions between precursors take place during the warmer months of the year when sunlight intensity is strongest. Conditions conducive to this type of pollution can occur up to a latitude of 60° North or the Arctic Circle (Nieboer et al., 1976). Hence given the proper mixture and amount of precursors, ozone levels can be expected to be elevated at some time of the year throughout most of Canada. Because of the relatively long life-time of ozone in the absence of high levels of NO_x, the pollutant can be transported over long distances.

The photochemical production of oxidants in the atmosphere is a complex function of the relationships among the concentrations of its precursors, sunlight intensity, meteorological conditions etc. The following sequence is commonly observed in urban areas (Haagen-Smit and Wayne, 1976). During the predawn hours of the morning when city activity is at a minimum, concentrations of NO and hydrocarbons slowly build up in calm wind conditions while ozone and NO_2 remain at low levels in the absence of photochemical reactions. After dawn with increasing urban activity NO and hydrocarbons increase and NO_2 begins to be generated by photolysis. After a couple of more hours, NO is reduced to low levels because it has been converted to NO_2, and NO_2 reaches its highest concentration. Ozone begins to accumulate until it reaches maximum concentration sometime after noon, and then gradually declines throughout the rest of the day. The portrait of events neglects the effects of any transport over the city from distant sources of ozone and its precursors. The variation of ozone and its precursors at a monitoring location in Vancouver illustrates the above discussion (Figure 2.2-1).

The basic reactions for production of ozone were given in Section 2.2.3 on nitrogen oxides. Here, it is necessary to point out that when reactive hydrocarbons are present with nitrogen oxides, they can form peroxy radicals that oxidize the nitric oxide, pumping it directly to nitrogen dioxide (Figure 2.2-2). This leaves very little of the NO to react with ozone, so that ozone builds up to large concentrations. The chain of reactions is stopped when radicals react with NO_2 to form stable products such as PAN by:

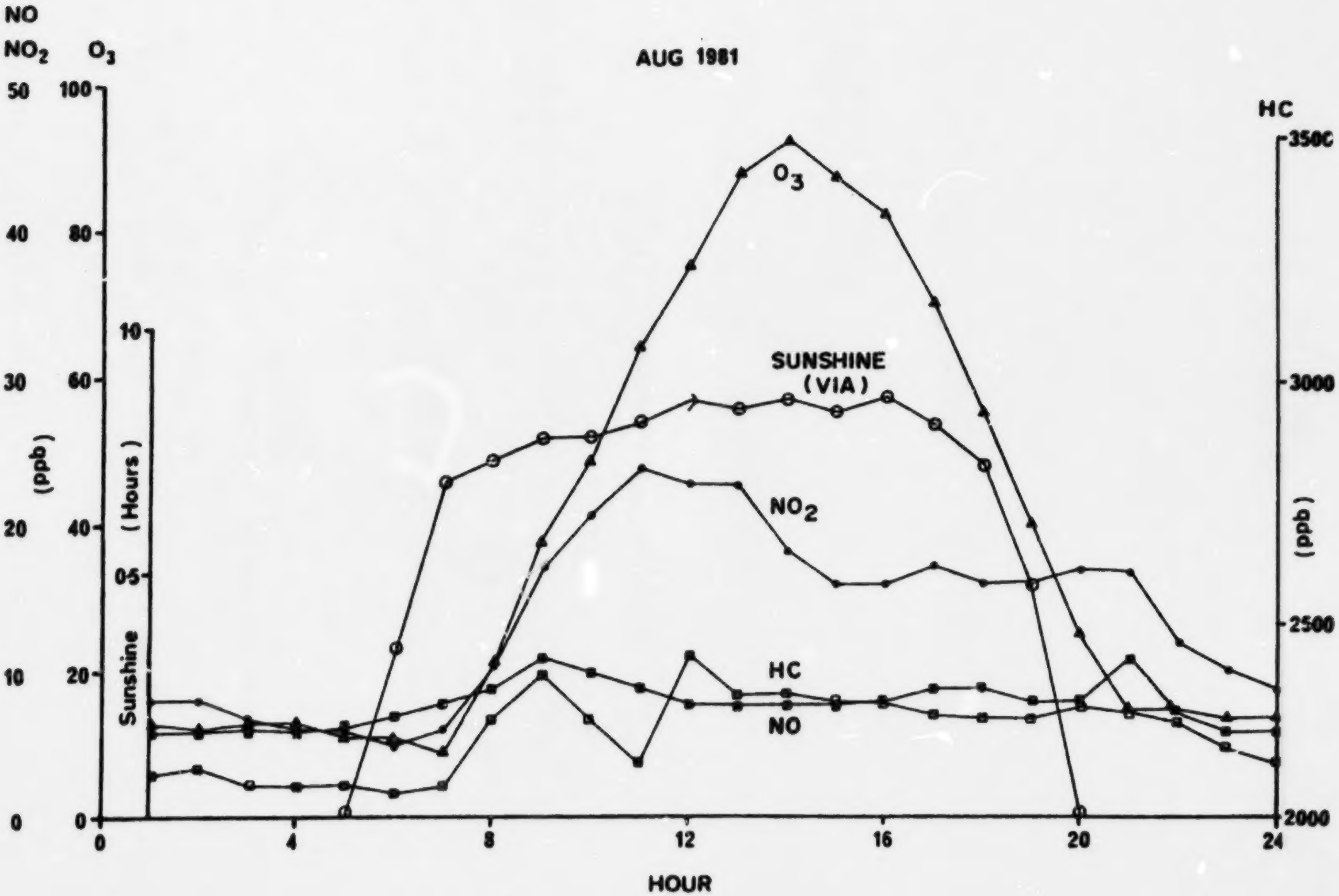
$$R\overset{\overset{O}{\|}}{C}O_2 + NO_2 \longrightarrow R\overset{\overset{O}{\|}}{C}ONO_2 \qquad (7)$$

Another oxidant, pernitric acid (HO_2NO_2), much like PAN, may be present in concentrations similar to PAN.

On occasions, researchers have found a surface ozone concentration maximum downwind of large urban areas (Chung, 1977) implying that the time for photochemical production of O_3 from NO_x and hydrocarbons requires a few hours. Graedel and Schiavone (1981) concluded from their modelling of the urban atmosphere that significant portions of city emissions are transported downwind where they are available for chemical reaction (or deposition).

FIGURE 2.2-1

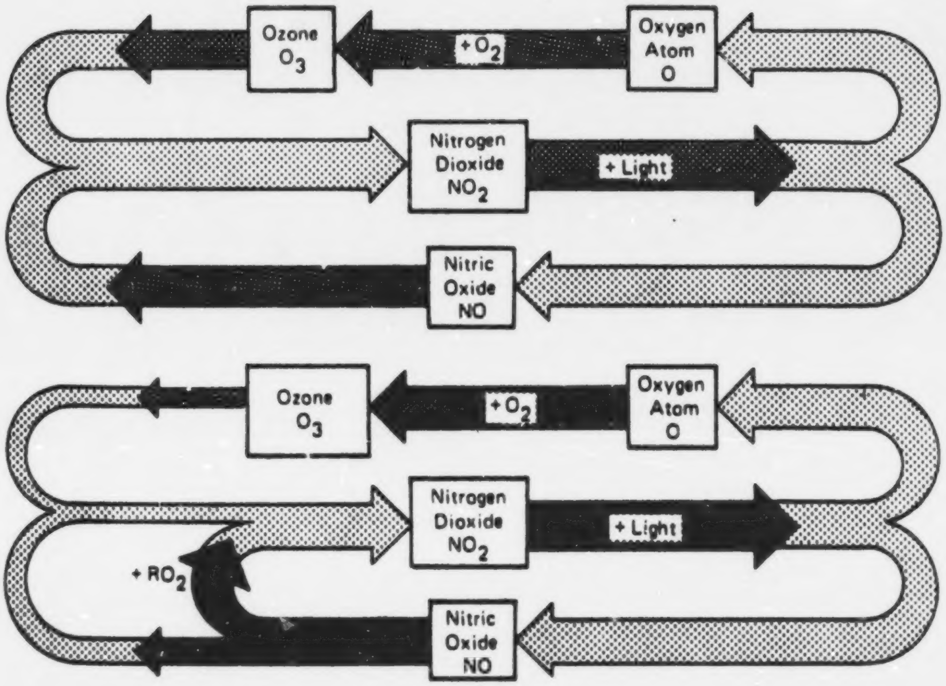
VARIATION OF OZONE AND ITS PRECURSORS IN VANCOUVER *



* Reproduced from Concord Scientific (1982)

FIGURE 2.2-2

THE PHOTOCHEMICAL CYCLE *


Ozone
O_3
$+O_2$
Oxygen
Atom
O
Nitrogen
Dioxide
NO_2
+ Light
Nitric
Oxide
NO
Ozone
O_3
$+O_2$
Oxygen
Atom
O
Nitrogen
Dioxide
NO_2
+ Light
$+RO_2$
Nitric
Oxide
NO

The NO-NO_2-O_3 cycle in air contaminated with NO_x only (above) and with NO_x and hydrocarbons (below).

* Reproduced from the National Academy of Sciences (1976)

There is a general consensus that reducing hydrocarbon emissions would reduce the production of photochemical oxidants (OECD, 1979). There is substantial controversy on the role of NO_x. An experimental model developed by Glasson (1981) suggests that hydrocarbon reduction reduces O_3 in urban as well as downwind areas while NO_x reduction increases O_3 and has little effect on O_3 in downwind areas. The results also indicate that both hydrocarbon and NO_x reduction will reduce NO_2 levels, with NO_x reduction being more pronounced. This study pertains specifically to Los Angeles and is not necessarily transportable to other urban centres. However, work conducted by several research groups with different modelling tools in different areas tends to predict that HC control alone is usually more efficient than combined HC and NO_x control and that, in addition, NO_x control alone may often increase the downwind O_3 burden (Hov and Derwent, 1981; Jones et al. 1983).

Over the past decade, a number of studies have pointed to the importance of long range transport in determining ozone levels in parts of Canada (Munn, 1973; Chung, 1977; Mukammel et al. 1982). During prolonged periods of air stagnation, ozone may be trapped in the lower atmosphere resulting in the gradual build-up of concentrations similar in levels to those observed in urban areas. Wolff and Lioy (1980) have documented an "ozone river" of high concentration which stretches from the northwest coast of the Gulf of Mexico to the lower Great Lakes and on to New England in the presence of warm tropical air during summer. Under these conditions, which are conducive to photochemical O_3 formation, even emissions from moderate size cities (populations ∿ 100,000) may contribute to the downwind ozone burden (Spicer et al. 1982). During such episodes scavenging of ozone in urban centres has been noted due to higher levels of nitric oxides in cities than in rural areas (Stasiuk and Coffey, 1974) resulting in lower concentrations in urban areas than in rural areas (cf. Equation 1 Section 2.2.3).

2.2.6 Diesel Exhaust Particulates

Comparisons of diesel-powered automobile emissions with its gasoline counterpart generally show much higher concentrations of particulates, higher PAHs, higher sulphates but lower carbon monoxide and total hydrocarbons. PAHs and sulphates are associated with the particulate emissions. A list of identified PAHs in diesel exhaust is given in Table 2.2-2. Light-duty catalyst gasoline cars emit particulates at a rate of about 0.05 g/km compared with 0.2-0.6 g/km for diesels (Baines et al. 1982). Approximately three-quarters of the particulate matter emitted from diesels is soot (National Research Council, 1981).

Airborne particles ranging in size from a few nanometers (10^{-9} metres) to perhaps 100 microns are ubiquitious components of the atmosphere. These particles, taken collectively, can contain every compound existing or produced on the earth that is solid or liquid at ambient conditions as well as sorbed traces of most gases. There is strong evidence that the atmospheric aerosol is composed of two distinct populations, substances having a diameter below 2 μm (fine particles) and particles produced by mechanical processes with diameters above 2 μm (coarse particles). The particle emissions of diesel engines belong to the fine particle class.

Ambient concentrations of particulates have commonly been measured in North America with a high-volume sampler. This instrument collects all suspended particulate matter up to a size of about 100 μm in diameter. Since the mass of the coarse particle fraction varies considerably and is much larger than the fine particle fraction, the measurement of total suspended particulates by high volume samplers bears little resemblance to the concentrations of fine particles (Dann, 1983).

TABLE 2.2-2

POLYCYCLIC AROMATIC HYDROCARBON (PAH) COMPOUNDS IN DIESEL EXHAUST PARTICULATES

Nonpolar PAH Identified in Diesel Exhaust Particulate Extract

dibenzothiophene
anthracene and phenanthrene
methyl dibenzothiophene isomers(3)
methyl (phenanthrene and anthracene) isomers(4)
dimethyl dibenzothiophene isomers(7)
dimethyl (phenanthrene and anthracene) isomers(13)
fluoranthene and pyrene
trimethyl dibenzothiophene isomers(9)
BaP, BeP, perylene,and isomers(3)
trimethyl (phenanthrene and anthracene) isomers(15)
tetramethyl dibenzothiophene isomers(12)
tetramethyl (phenanthrene and anthracene) isomers(16)
benzo(g,h,i) fluoranthene
benz(a)anthracene, chrysene, benzo(c)phenanthrene, triphenylene isomers(2)
methyl benz(a)anthracene isomers(4)
pentamethyl dibenzothiophene isomers(4)
dimethyl benz(a)anthracene isomers(2)
methyl (fluoranthene and pyrene)isomers(7)

Moderately Polar PAH Derivatives Identified in Diesel Exhaust Particulate Extract

benz(a)anthracenedione
methyl (anthrone and phenanthrone) isomers
thiozanthone isomer
dimethyl (anthrone and phenanthrone) isomers
pyrenone
trimethyl (anthrone and phenanthrone) isomers
methyl thioxanthone
dimethyl thioxanthone isomers(2)
benz(d,e)anthrone and isomers(3)
1-nitropyrene
1,1' biphenyl-ol
9-fluorenone
(pyrene or fluoranthene) carboxaldehyde
dibenzofuran carboxaldehyde
phenanthrone
anthrone isomer
9-xanthone
xanthene carboxaldehyde
(anthracene or phenanthrene)dione
dibenzothiophene carboxaldehyde
methyl (anthracene or phenanthrene)dione
phenanthrene carboxaldehyde
anthracene carboxaldehyde
methyl (anthracene and phenanthrene) carboxaldehyde isomers(9)
dimethyl (anthracene and phenanthrene) carboxaldehyde isomers(8)

Reproduced from Prater _et al_. 1981

2.2.7 Acid Precipitation

The term acid rain was first used in 1872 in a publication entitled "Air and Rain.The Beginnings of a Chemical Climatology" (R.A. Smith, 1872). This publication enunicated many of the principal ideas that are now part of the present understanding of the acid rain phenomenon. Contemporary concepts about acid precipitation and its environmental effects originated in three fields of science: limnology (study of lakes), agriculture, and atmospheric chemistry. The science of atmospheric chemistry began in Sweden during the 1950's; the first study of the causes of acid precipitation and its impact on aquatic ecosystems began during the same period; and in agriculture, studies commenced as early as the late 19th century. Through various steps over the last century the concepts of acid precipitation have been transported from the domain of scientific curiosity to that of public concern.

Acidity in a solution such as rain is synonymous with the presence of hydrogen ions (H^+) and is commonly expressed by a pH value. This measure of acidity is the negative logarithm of the hydrogen ion concentration ($pH = -\log (H^+)$). In pure water the solution is neutral and has a pH of 7. The pH will become less than 7 when the solution becomes acidic. Since the pH is a logarithm, solutions of pH 6, 5 and 4 contain respectively 1, 10, and 100 microequivalents of acidity (H^+) per liter. In other words a liquid having a pH of 4 will be 100 times as acidic as one with pH 6.

Carbon dioxide contained in the atmosphere dissolves in rain water to a certain extent and forms carbonic acid. At normal concentrations of CO_2 in the atmosphere, rainwater saturated with carbonic acid has a pH of about 5.6. Other substances reaching the atmosphere shift the pH one way or another and determine the chemistry of natural precipitation. In areas where the soil is alkaline or basic, the incorporation of wind eroded dust in precipitation may elevate pH levels to 6 or 7. Precipitation in coastal areas has enhanced concentrations of such sea-spray components as sodium (Na) and chloride (Cl). Gases such as sulphur dioxide, hydrogen sulphide, and nitrogen oxides which come from natural sources, can also alter the chemistry of precipitation. Sulphur dioxide and hydrogen sulphide are oxidized and hydrolyzed in the atmosphere to sulphuric acid while nitrogen oxides are similarly converted into nitric acid. If these acids are present in significant quantities, they can acidify precipitation below pH 5.6. Recent examination of the composition of precipitation in remote areas of the world suggests that the lower limit of the natural mean pH of precipitation is probably equal to or greater than 5 (Galloway et al., 1982). Acid precipitation is commonly regarded as rain or snow which has a pH of 5.6 or lower.

Various measurements of the chemical composition of precipitation indicate that acids are present in larger quantities than would occur naturally. This increased acidity is usually attributed to SO_2 and NO_x pollution of the air; of direct relevance to this study is the importance of the NO_x contributions. Acid precipitation has been well documented as a regional phenomenon in eastern Canada (Whelpdale, 1980), Scandinavia (Likens et al., 1979) and eastern United States (ibid, 1979). The chemistry of precipitation portrayed for four areas in Canada in terms of the concentration of major ions is given in Table 2.2-3. Hydrogen, sulphate (SO_4), nitrate (NO_3) and chloride contribute to the acidity; the other ions tend to neutralize an aqueous solution.

The precipitation for the three stations in eastern Canada, which may be classified as acidic have high levels of sulphate and nitrate compared with that of Port Hardy. The two stations near the oceans (Kejimkujik and Port Hardy) have high concentrations of chloride but this ion, to a great extent, is neutralized by the high levels of sodium. Both ions originate primarily from sea salt. Some sort of sulphate at these locations may also be attributed to sea salt.

Maps of the spatial distribution of pH and the most important chemical species in precipitation over North America have been prepared by the Atmospheric Environment Service of Environment Canada (Barrie and Sirois, 1982). The pH maps (Figure 2.2-3) show a zone of depressed pH coincident with the greatest sulphate and nitrate deposition.

TABLE 2.2-3

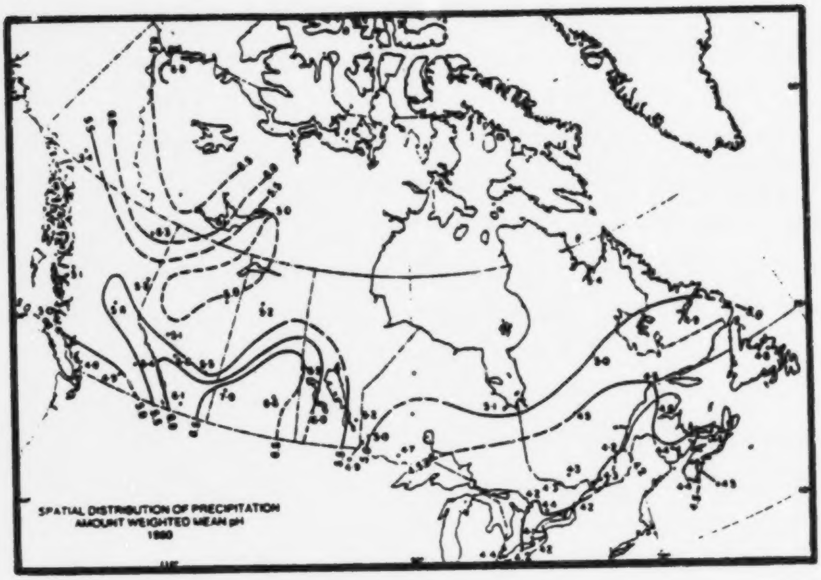
SELECTED EXAMPLE OF PRECIPITATION CHEMISTRY IN CANADA FOR 1980

	PORT HARDY BRITISH COLUMBIA pH 5.0	DORSET ONTARIO pH 4.2	MANIWAKI QUEBEC pH 4.3	KEJIMKUJIK NOVA SCOTIA pH 4.5
Hydrogen (H^+)	10.1	64.5	49.1	30.1
Sulphate ($SO_4^=$)	10.0	31.0	31.0	17.0
Nitrate (NO_3^-)	2.6	32.2	30.1	11.0
Ammonium (NH_4^+)	2.6	23.4	32.4	3.3
Chloride (CL-)	61.7	6.2	4.3	35.6
Calcium (Ca++)	5.3	8.2	8.9	7.4
Magnesium (Mg++)	6.0	2.0	2.0	4.6
Potassium (K+)	4.0	2.0	2.7	2.4
Sodium (Na+)	46.0	1.8	6.3	33.7

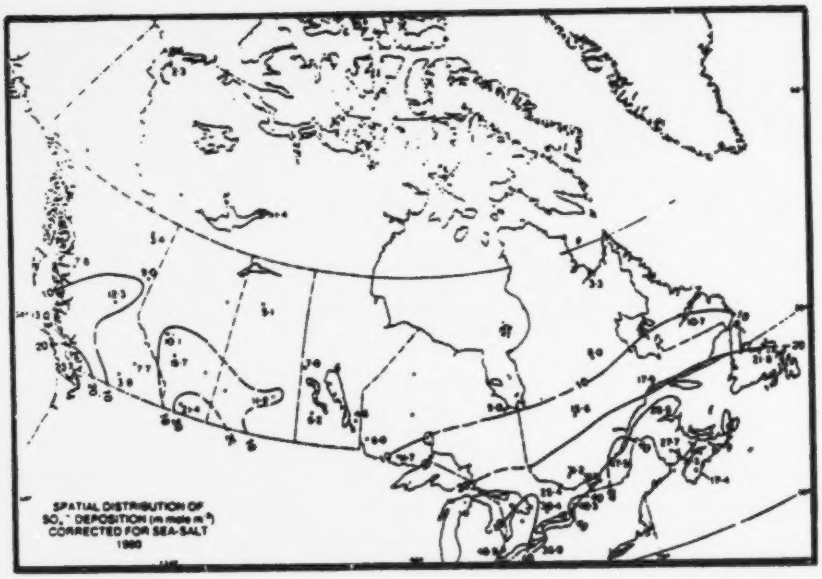
Chemistry of precipitation is portrayed in terms of micromoles per liter of 9 ions.

Source: Barrie and Sirois (1982)

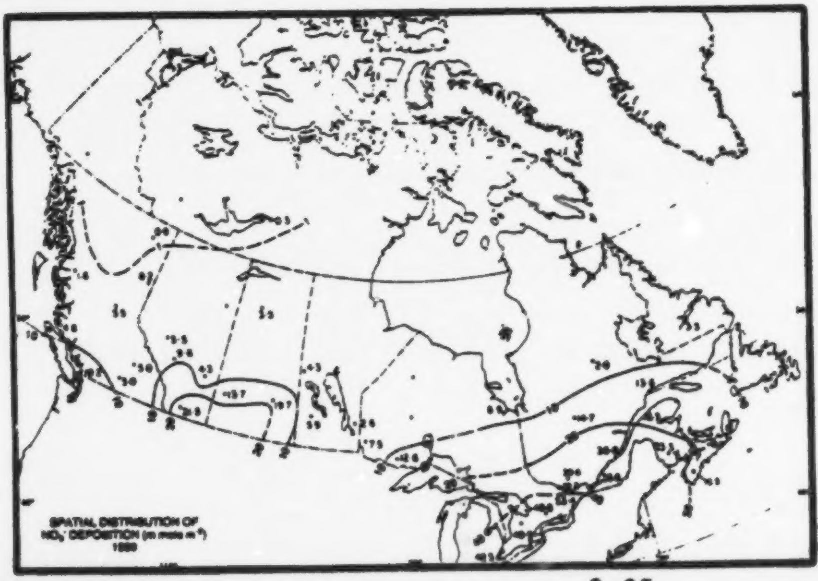
FIGURE 2.2-3
WET DEPOSITION


SPATIAL DISTRIBUTION OF PRECIPITATION
AMOUNT WEIGHTED MEAN pH
1980

A) Precipitation amount-weighted mean annual pH in Canada for the calendar year 1980


SPATIAL DISTRIBUTION OF
SO4= DEPOSITION (m mole m-2)
CORRECTED FOR SEA-SALT
1980

B) Precipitation amount-weighted mean sulfate ion deposition for 1980
(m moles per square metre)
(0.961 kg/ha=1m mole/m²)


SPATIAL DISTRIBUTION OF
NO3- DEPOSITION (m mole m-2)
1980

C) Precipitation amount-weighted mean nitrate ion deposition for 1980
(m moles per square metre)
(0.62 kg/ha=1m mole/m²)

LEGEND: · CANSAP
● APN

Source:
Barrie and Sirois (1982)

The major pathways leading to acidification of the air, suspended particles, dews, fog droplets, cloud nuclei, and raindrops are shown in Figure 2.2-4. The formation of acids is linked to the photochemical reactions which produce radicals and oxidants. The relative importance of gaseous phase and aqueous-phase pathways for various geographical regions have not been established but there are indications that the aqueous-phase is dominant for H_2SO_4. The relative importance of the pathways for HNO_3 is not known, but it appears that the overall HNO_3 formation rate is greater than five times that for H_2SO_4. Some additional details on nitric acid are given later in this section. Provided that scavenging of H_2SO_4 and HNO_3 are not appreciably different, the faster transformation rate of NO_2 to nitric acid would indicate that nitrate in precipitation would be of a more local origin than sulphate. It should be noted that either oxidation scheme leads to eventual deposition in a wet or dry form.

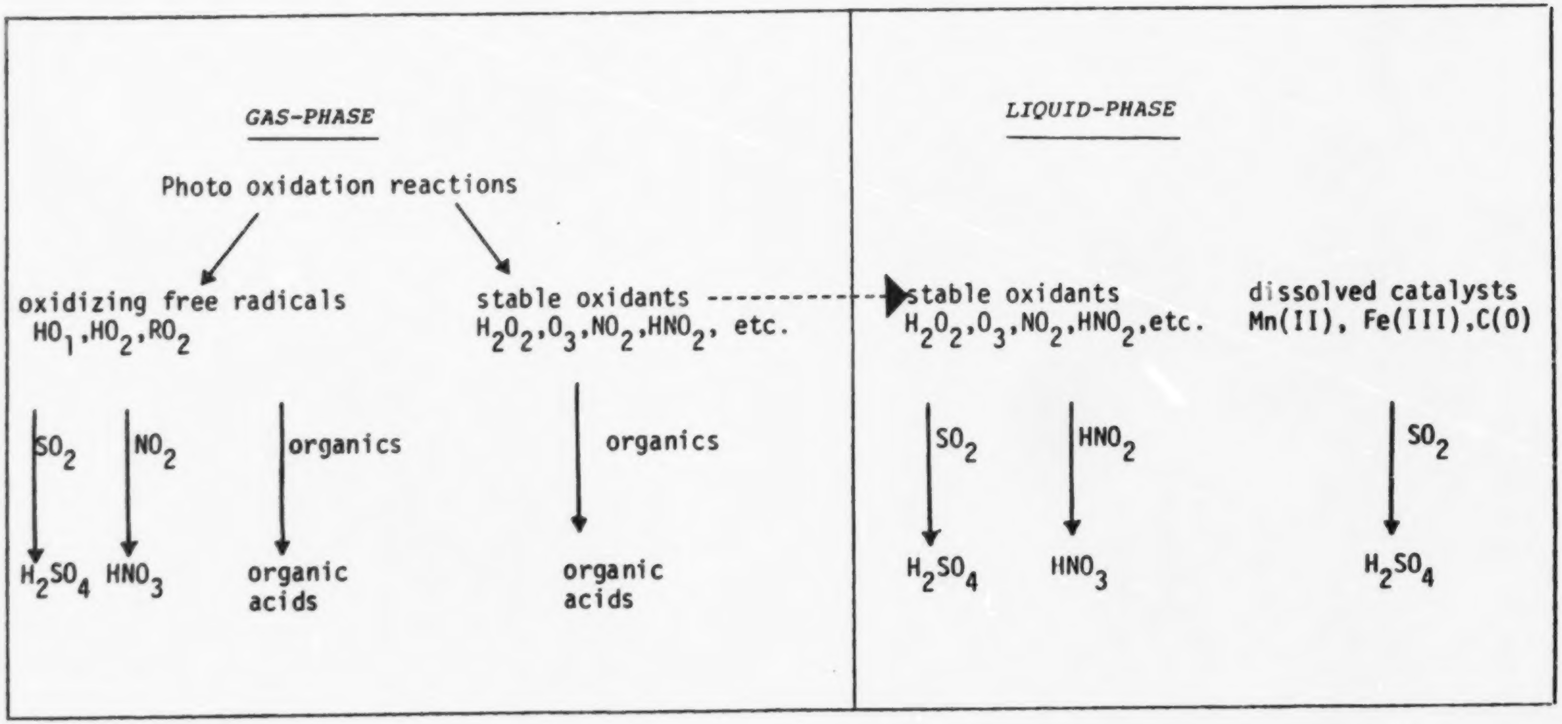
The Linearity Debate

There is considerable debate on the interaction of radicals and oxidants in the formation of sulphuric acid, one of the major components of acid precipitation. Rodhe et al. (1981) presented evidence that NO_x tends to reduce levels of OH and H_2O close to a source area thereby delaying and decreasing the transformation of SO_2 to H_2SO_4. This is caused mainly because of the common dependence of SO_2 and NO_x concentration on the OH radical. When dealing with a coupled system like the one studied by Rodhe et al. (1981) one may not assume a proportional dependence of concentrations on the emission ratio i.e. reactions are non-linear. However, the model possesses serious deficiencies which limit the usefulness of its predictions (MOI, 1982).

The reactions of gas- and aqueous-phase are non-linear mainly because free radical concentrations and oxidants are not constant over time and space. The MOI document (1982) listed the following conclusions with respect to the acidification of precipitation and linearity:

FIGURE 2.2-4

MAJOR PATHWAYS FOR ACIDIFICATION IN THE ATMOSPHERE


GAS-PHASE
Photo oxidation reactions
oxidizing free radicals
HO_1, HO_2, RO_2
stable oxidants
H_2O_2, O_3, NO_2, HNO_2, etc.
SO_2
NO_2
organics
organics
H_2SO_4
HNO_3
organic acids
organic acids
LIQUID-PHASE
stable oxidants
H_2O_2, O_3, NO_2, HNO_2, etc.
dissolved catalysts
Mn(II), Fe(III), C(0)
SO_2
HNO_2
SO_2
H_2SO_4
HNO_3
H_2SO_4

Reproduced from MOI, 1982

gas-phase:

(i) reaction rates are non-linear with regard to SO_2 because free radical concentrations are not constant over time and space

(ii) gas-phase rates are first order in SO_2, therefore a reduction in SO_2 concentration will result in direct reduction of H_2SO_4 if free radical concentrations are constant

(iii) rates are first order in free radical concentrations; if there is reduction in free radical concentration (through oxidant precursor control) there will be a corresponding reduction in H_2SO_4 formed

aqueous-phase:

(i) reaction rates are non-linear in regard to SO_2 because atmospheric liquid water content is not constant over time and space

(ii) rates are first order in SO_2 in the aqueous phase

The Work Group recognized that although non-linear microscale events apply to individual events of less than one day, it was possible that long term averages (monthly or greater) derived from linear models might give similar results to non-linear models. Two selected examples of non-linearity in microscale are now discussed.

The reaction of NO_x with SO_2 in aqueous aerosols, is an example of the type of microscale processes that may occur. Schryer _et_ _al_. (1983) presented experimental data indicating that significantly greater sulphate yields are obtained when NO_2 is present along with a catalyst than when it is not. The catalyst in this case was carbon (soot). However NO_2 is practically insoluble in water. Consequently, the relative importance of this mechanism in overall acidification of precipitation may not be great.

Additionally, recent evidence from studies conducted in northwest Indiana suggests that organic nitrogen compounds such as peroxyacetylnitrate (PAN) are prominent in the atmosphere, are soluble in rainwater, and can affect rain acidity (Holdren et al. 1982). The average concentration of PAN was about 2.5 mg/m^3 (½ ppb). These levels are comparable to the amount of HNO_3 found at the same location. Results of PAN-water interactions show that PAN is soluble in rainwater and gradually decays in solution to nitric acid and one or more organic products. The results also indicate that PAN can also affect rainfall acidity by serving as an oxidizing agent for gases such as sulphur dioxide and nitrogen dioxide. It may convert them to their respective strong acids, sulphuric and nitric, in cloud droplets thus indirectly affecting acid precipitation.

Switching from microscale to long-term macroscale processes, recent studies by Oppenheimer (1983 a,b,c) suggest that long-term averages of acid (sulphate) deposition can be adequately described by a linear model. The combined results of his studies indicate that the relationship of SO_2 emissions in the eastern U.S. to annual wet sulphur deposition is nearly linear in a regionally averaged sense for uniform emissions reductions larger than about 10 percent; furthermore, the formation rate of aerosol sulphate is proportional to SO_2 concentrations on more local scales. Since emitted SO_2 which passes through precipitation is essentially fully oxidized during wet removal, according to the approach of Oppenheimer, concerns that reductions in co-pollutants may indirectly increase wet sulphate deposition by altering intermediate oxidant concentrations (i.e. H_2O_2, O_3, NO_2 etc) are potentially without basis.

All of the conversion processes of SO_2 and NO_2 to acids and aerosols require oxidizing agents such as hydrogen peroxide, ozone, and hydroxyl radicals (e.g. Penkett 1979, cf. Figure 2.2-4). The production of these chemicals is directly related to reactive hydrocarbon precursors and in some cases to NO_x. In microscale processes, the chemical conversions of airborne pollutants all occur at different rates depending on the amounts of SO_2, NO_x, and reactive hydrocarbons emitted. Thus the relationships between SO_2 and NO_x emissions and sulphuric and nitric acid deposition is hard to estimate for events. However, there is an indication that on regional scales and over long averaging times that sulphate deposition is proportional to SO_2 emissions. No such evidence has yet been given for nitrate deposition although the following investigations tend to indicate the possibility of linearity.

Likens (1972) first indicated that nitric acid, resulting from the transformation of NO_x, adds to the acidity of precipitation in the eastern United States. Brosset et al. (1975) in Sweden showed that gaseous nitric acid increases acid deposition. Although sulphuric acid has been found to be the dominant source of acidity in Canada, the United States, and Europe, nitric acid accounts for almost one-third of all acid deposition and there is evidence that this fraction is rising (Galloway and Likens, 1981). The maximum contribution of the acidity of wet deposition of NO_3^- and $SO_4^=$ can be calculated by comparing their concentrations (in µeq/l) to that of H^+. In southern Ontario the maximum possible contribution of HNO_3 and H_2SO_4 to the acidity of wet deposition was 39% and 87% (Schneider et al. 1979).

Galloway and Dillon (1982), using continent wide data, indicated that for the western half of North America, there is enough NO_3 to account for all the acidity of wet deposition. In a large region of eastern North America, NO_3 can only account for ≤50% of the acidity. In the case of H_2SO_4, there is enough SO_4 to account for all of the acidity of wet deposition in Canada. While the absolute contribution of HNO_3 is uncertain, its levels appear to be increasing relative to sulphuric acid, in proportion to increased NO_x emissions.

For mobile sources, consideration must be given to the probability that a significant fraction of NO_x emissions are removed by dry deposition in the vicinity of sources (e.g. cities and highways). The ratio of wet to dry deposition increases with increasing distance from the source and precipitation provides the major removal process in remote areas (Logan, 1982).

Until recently, there was some debate over the natural versus anthropogenic strengths of NO_x. Logan (1982) has provided evidence that anthropogenic sources supply more NO_x to the atmosphere in Canada and the United States than natural sources (Table 2.2-4). Some additional considerations of the relative strengths of emissions has previously been discussed in Section 2.2.3.

There is one final aspect regarding the relative importance of nitric acid. Recently, fog has been investigated as a new aspect of the acid deposition phenomenon (Wisniewski, 1982, Waldman et al., 1982). Highly acid fogs and drizzle (pH 2-3.5) have been documented in Japan during episodes which have occurred with a frequency of about 1 per year and are mainly attributed to NO_x emissions. The attention being directed to acid fog is due to its far higher acidity than has been reported for rainwater. In California urban areas, it was found that most of the studies of acid precipitation have been centred on the role of sulphuric acid. However, the ratio of NO_3 to SO_4 in fog water was quite different from that in rainwater. The ratio on an equivalence basis in fog mirrored the emissions ratio of NO_x to SO_x (2.5:1) while in rainwater it was less than 1.

Rhodes and Middleton (1983) summarize aspects of the relationship between acid rain and mobile sources of NO_x: "Because increased reduction in mobile sources is not sufficiently recognized as part of the current acid rain control strategies, if mobile emissions continue to increase, this oversight could result in no overall reduction in total acid in some locations, just a shifting of the relative contributions by sulphuric and nitric acids. Also automobiles can transport acid precursors directly to sensitive

TABLE 2.2-4

REGIONAL NO_x BUDGETS FOR NORTH AMERICA

(UNITS: 10^{12} gm N yr^{-1})

	U.S.	Canada	N. America	Eastern N. America[b]
Sources				
Fossil fuel combustion	5.8	0.6	6.4	3.5
Others	~0.8	~0.3	~1.1	~0.4
Total	6.6	0.9	7.5	3.9
Sinks				
Wet deposition	1.5	0.9	2.4	1.5
Dry deposition	1.4-2.7	0.35-0.7	1.7-3.4	1.0-2.0
Total	2.9-4.2	1.2-1.6	4.1-5.8	2.5-3.5
Export[c]			1.7-3.4	0.4-1.4

after Logan (1982)

regions, without the dilution of pollutants through long-range transport." It should be emphasized that the above is a statement of concern and cannot be validated with the current state of knowledge. The chemistry of nitrogen in the atmosphere is far less quantified than sulphur.

3. AIR QUALITY CRITERIA AND STANDARDS

3.1 What are Air Quality Criteria and Standards?

The term Air Quality criteria as used in this report, refers to observed effects or responses of one or several atmospheric trace substances on a defined receptor or population under specified conditions. This approach to elaborating air quality criteria is similar to that adopted by the World Health Organization (WHO), the Environmental Protection Agency (EPA) in the United States, the United Nations 1972 Stockholm Conference on the Human Environment and the National Research Council of Canada. Under this approach, the term "adverse effects" is not used. Criteria are based on solely scientific data, and entail no value judgement such as an identification of "beneficial" or "adverse" effects.

Standards prescribe an air pollutant value that cannot legally be exceeded during a specific time in a given political jurisdiction. These are arbitrary man-made laws, in contrast to criteria which are observed facts. There are two major types of air standards: ambient air quality standards which apply to concentrations of pollutants in outdoor air at the receptor, and emission standards which apply at the point of emission. These two categories of standards are interrelated. An emission standard in many cases is set to ensure compliance with an ambient air standard. In Canada, ambient air quality objectives are used rather than ambient air quality standards. The main distinction between objectives and standards is that the former are not legally enforceable. The establishment of objectives requires the same process of interpretation of scientific criteria as does the setting of standards.

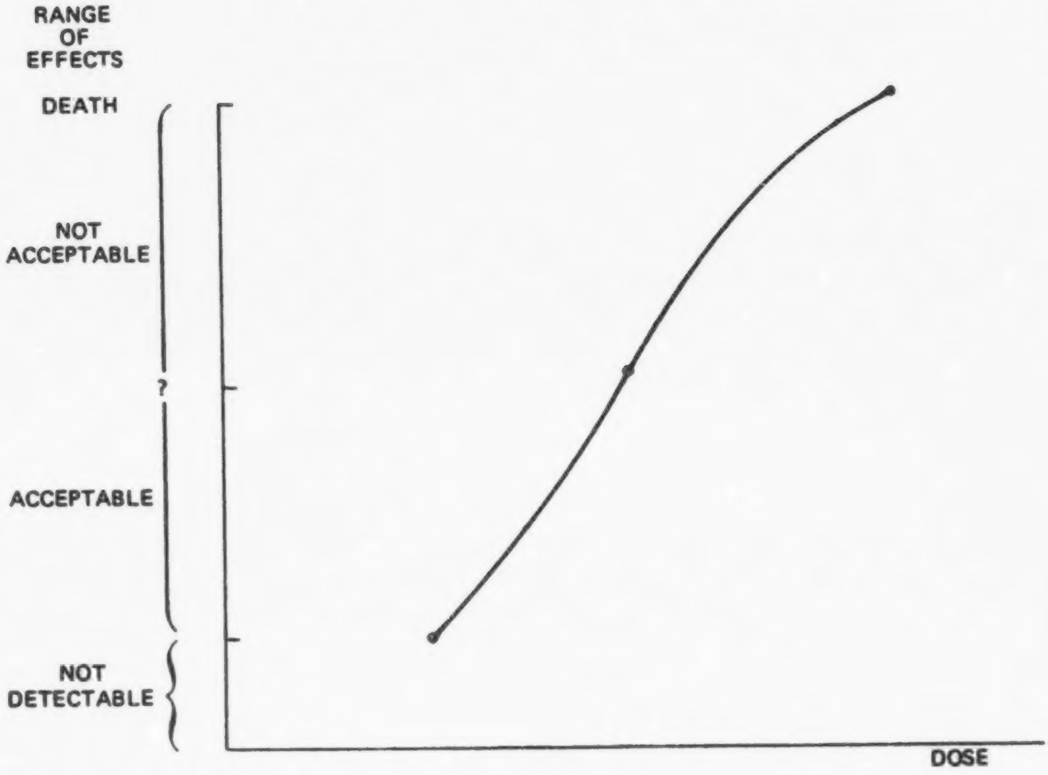
In order to set an ambient air quality standard, criteria are needed relating amounts of pollutant to observed effects on a sensitive receptor. The ideal situation for the setting of standards is documentation encompassing a wide range of pollutant concentrations and effects. The distinction between acceptable and unacceptable effects is a value judgement based on social, political, technical or aesthetic factors and is the responsibility of a control agency. The concentration chosen to distinguish between acceptable and unacceptable becomes a standard when legally proclaimed.

Criteria and dose-response relationships are synonymous. The dose-response relationship incorporates the quantification of effects versus the dose (concentration over a period of time) of a substance (Figure 3.1-1). Data tend to suggest that there is a continuum of effects ranging from severe and readily identifiable effects at high concentration levels to subtle and uncertain effects at very low concentrations. Literature search, critical review, and extraction of pertinent information results in the production of criteria documents. Knowledge of dose-response relationships is continually evolving so that criteria need periodic revision.

This current report is in effect a criteria document specifically aimed at providing dose-response relationships for the atmospheric pollutants produced by automobiles. The primary methodology used to produce this report was the review of criteria documents (produced by various countries and agencies) for relevant pollutants. Secondarily, a literature search was conducted to incorporate recent information that became available subsequent to the publication of criteria. Air quality standards are also examined and represent the influence of social, economic and judicial considerations applied to scientific data and interpretations of what are adverse effects. These standards accordingly represent a wide range of prescribed levels for each pollutant, although, in all cases, the primary purpose is to protect public health and welfare against adverse air pollution effects.

FIGURE 3.1-1

DOSE OF AGENT VERSUS EFFECTS CURVE*



*Adapted from Sanderson, 1978.

As an introduction to various countries' approaches in documenting criter and establishing standards, a review of the approach used by the World Health Organization in assessing effects is now presented. The results of investigations by this international body are often used and sometimes adopted by various countries as the basis of standard promulgation.

In 1972, the World Health Organization (WHO) initiated an Environmental Health Criteria programme to assess the health effects of pollution. The major objectives of the programme were:

. to assess existing information on the relationship between exposure to environmental pollutants and human health, and

. to provide guidelines for setting exposure limits consistant with the protection of public health.

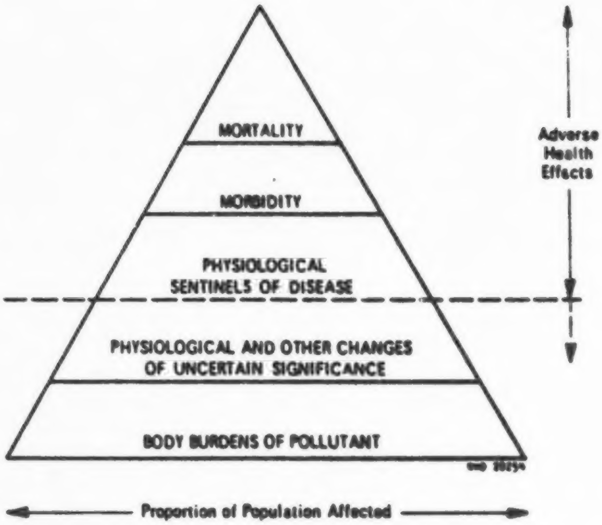
In 1980, this program was incorporated into a more comprehensive International Programme on Chemical Safety. Sponsored by the WHO, the United Nations Environment Programme (UNEP), and the International Labour Organization, this program's major objectives are those of the previous WHO program.

The WHO has outlined the basic concepts of the administrative use of air quality criteria and guides (WHO, 1972). The first consideration is the definition of an adverse health effect. Death and disease represent the extreme end of a spectrum of response (Figure 3.1-2). In addition, within an exposed population, there may be groups especially sensitive to exposure. The ideal situation is to have a complete set of dose-response curves for different effects and for different types of populations exposed. The WHO defines four levels of effects:

Level I	Concentration and exposure time at or below which, according to present knowledge, neither direct or indirect effects (including alteration of reflexes or of adaptive or protective reactions) have been observed.
Level II	Concentrations and exposure times at and above which there is likely to be irritation of organs, harmful effects on vegetation, visibility reduction, or other adverse effects on the environment.

FIGURE 3.1-2

SCHEMATIC SPECTRUM OF BIOLOGICAL RESPONSE TO POLLUTANT EXPOSURE


MORTALITY
MORBIDITY
PHYSIOLOGICAL SENTINELS OF DISEASE
PHYSIOLOGICAL AND OTHER CHANGES OF UNCERTAIN SIGNIFICANCE
BODY BURDENS OF POLLUTANT
Adverse Health Effects
Proportion of Population Affected

Based on a diagram in United States Congress Document No. 92-241, 1972.

Reproduced from WHO, 1972.

Level III Concentrations and exposure times at and above which there is likely to be impairment of vital physiological function or changes that may lead to chronic diseases or shortening of life.

Level IV Concentrations and exposure times at and above which there is likely to be acute illness or death in susceptible groups of the population.

Since there is much uncertainty in the dose-response relationships, and Levels I and II are not easily distinguished, a safety factor is commonly used to be prudent. The magnitude of a safety factor can be based on:

(i) cost-benefit analysis
(ii) scientific uncertainties
(iii) protection from a specific effect
(iv) risk analysis

Some scientific uncertainties in defining air quality criteria (Munn, 1978) are:

(a) Receptor response has inherent variability even in a controlled laboratory setting.
(b) Receptor response is not reproducible, due to adaptation.
(c) Receptor response depends on age, stage of development and health of the organism.
(d) The response of a receptor sometimes does not occur until a few hours to a few years after the dose has been received. For genetic effects, the establishment of air quality criteria is almost impossible.
(e) The dose sometimes reaches the receptor through multiple routes, e.g. through inhalation, drinking water,etc.
(f) Laboratory results are sometimes qualitatively and frequently quantitatively different from outdoor results. (The responses of

animals are different from those of humans; the microclimate in greenhouses is different from that outdoors; etc.)

(g) The outdoor environment has substantial variability not only in the concentrations of trace substances, but also in the associated factors/processes (e.g. temperature, humidity, sunlight).

(h) The outdoor environment is variable in space as well as in time. Humans move from place to place, and spend considerable periods indoors. Population effects/responses therefore are not easily related to measurements of air quality made at fixed monitoring points.

(i) Air quality criteria are usually based on atmospheric concentrations measured at fixed points. Yet for vegetation, effects/responses depend on uptake rates rather than on air concentrations.

(j) The question of thresholds (i.e. WHO Level I) in dose-response relationships needs careful investigation. The existence or non-existence of a threshold has a profound effect on air pollution control strategies.

One aspect of defining standards or objectives is stating one or more pollutant concentration averaging times. Averaging times usually range from ½ hour up to one year. Different averaging times may be required because the time pattern of pollutant levels can be a determining factor in distinguishing adverse effects. For example, the total dose of an air pollutant over a long period may be more important for one effect, while a short period dose may be relevant for another effect of the same substance.

Standards or objectives may vary from country to country and within a country over the course of time as new knowledge is acquired. The long-term goal is to protect against all effects relevant to human health, adopting a safety factor to ensure such protection. Consideration is also given to adverse effects on the environment such as vegetation damage and visibility degradation. The concept of a standard to protect receptors from significant harm is statistical, based on an aggregate of individual cases of significant harm. The adoption of a standard may not confer protection on every individual.

3.2 Canada's Approach

3.2.1 Federal Government

The federal Clean Air Act of 1971 provided for the formulation of national air quality objectives. These objectives are the goals toward which air pollution control programmes are oriented. Canada's National Air Quality Objectives (NAQO) do not have legal force unless they are utilized as standards by provincial governments. These objectives are designed to protect public health and welfare, hence the purpose of revising automotive standards would be to ensure objectives are not exceeded. Three levels of air quality objectives are specified based on the recognition that there are a number of threshold levels for each pollutant or combination of pollutants (Table 3.2-1).These varying thresholds are dependant not only on scientific criteria, but also upon the socio-economic and administrative concerns of the Federal government.

Two sub-committees on National Air Quality Objectives, one on acceptable and desirable levels and the other on tolerable levels work under the following terms of reference:

(a) to list air contaminants for which NAQO's should be prepared
(b) to evaluate the criteria
(c) to recommend on the basis of best available knowledge, the concentration time values of air contaminants for establishment of NAQO's.
(d) to report scientific findings to the Federal-Provincial Committee on Air Pollution in the form of recommendations

The sub-committees are comprised of medical and other scientific experts from governments (federal and provincial) and universities.

TABLE 3.2-1

DEFINITION OF THE THREE LEVELS OF CANADIAN NATIONAL AIR QUALITY OBJECTIVES

Maximum Desirable Level (comparable with WHO Level I*)	- ultimate goal for air quality - a basis for antidegradation policy for the unpolluted parts of the country and for the continuing development of control technology
Maximum Acceptable Level (comparable with WHO Level II)	- provides adequate protection against effects on soil, water, vegetation, materials, visibility, personal comfort and well-being - realistic objective today for all parts of Canada. When this level is exceeded, control action by a regulatory agency is indicated
Maximum Tolerable Level (roughly comparable to WHO Level III, while WHO Level IV is somewhat above it)	- denotes concentrations of air contaminants that require abatement without delay to avoid further deterioration of conditions to an air quality that endangers the prevailing Canadian lifestyle or ultimately, to an air quality that poses a substantial risk to public health

*see Section 3.1 for definition of WHO Levels

Carbon monoxide, oxidants (ozone), and nitrogen dioxide objectives have been promulgated. The background information supporting the maximum acceptable and desirable levels are found in a 1976 publication entitled "Criteria for National Air Quality Objectives." The Sub-committee on Desirable and Acceptable Air Quality Objectives is currently revising documentation for carbon monoxide and nitrogen dioxide (R.J. Powell, H.P. Sanderson, personal communication, 1983). The Sub-committee on Tolerable Air Quality Objectives is also preparing background information for tolerable objectives for all three pollutants utilizing a revised definition of a tolerable level (D. Hutchinson, personal communication, 1983). A maximum tolerable level is now considered to denote time-based concentrations of air contaminants beyond which, due to a diminishing margin of safety, appropriate action is required to protect the health of the general population (cf. Table 3.2-1). Draft documentation and possible air quality objective level revisions were not available for review at the time of preparation of this report.

In addition to the activities of the Federal-Provincial Committee on Air Pollution and its subcommittees, the National Research Council of Canada (NRCC) has established an Associate Committee on Scientific Criteria for Environmental Quality in response to a mandate provided by the Federal Government to develop scientific guidelines for defining the quality of the environment. Whereas the Subcommittees on Air Quality Objectives provide recommendations for objective levels, the NRCC is concerned strictly with scientific criteria.

3.2.2 Provincial Government

Air quality objectives, criteria and regulations of the federal and provincial governments, provided in Table 3.2-2, vary quite significantly.

TABLE 3.2-2

AIR QUALITY OBJECTIVES, CRITERIA AND REGULATIONS IN CANADA

Air Contaminant	Federal Objectives[a]			British Columbia Objectives	Alberta Regulations	Saskatchewan Regulations	Manitoba Objectives		Ontario Criteria	Quebec Regulations	New Brunswick Regulations	Nova Scotia Objectives		Newfoundland Criteria
	Desirable range	Acceptable range	Tolerable range				Maximum desirable	Maximum acceptable				Maximum desirable	Maximum acceptable	
Carbon Monoxide (mg/m^3)														
1 hour average	0–15	15–35	—	5.2–35.0	15	15	15	35	36.2	34	35	15	35	35
8 hour average	0–6	6–15	15–20	5.8–15.2	6	6	6	15	15.7	15	15	6	15	15
24 hour average	—	—	—	—	—	—	—	—	—	—	—	—	—	10
Oxidants (ozone) ($\mu g/m^3$)														
1 hour average	0–100	100–160	160–300	—	160	100	100	160	165	157	—	100	160	160
24 hour average	0–30	30–50	—	—	50	30	30	50	—	—	—	30	50	50
annual arithmetic mean	—	0–30	—	—	—	—	—	30	—	—	—	—	30	30
Nitrogen Dioxide ($\mu g/m^3$)														
1 hour average	—	0–400	400–1000	—	400	400	—	400	400	414	400	—	400	400
24 hour average	—	0–200	—	—	200	200	—	200	200	207	200	—	200	200
annual arithmetic mean	0–60	60–100	—	—	60	100	60	100	—	103	100	60	100	—

[a] Prince Edward Island has adopted the Federal desirable air quality objectives as guidelines.

Prince Edward Island: The province has adopted as a guideline the national desirable ambient air quality objective considering that the province is relatively free from air pollution problems (Lapointe, 1978).

Newfoundland: Air Pollution Control Regulations were promulgated in 1981, and, in most cases, they conform to national acceptable levels. Newfoundland's approach is comparable with that of Ontario and is discussed there.

Nova Scotia: The province uses the national ambient air quality objectives for the evaluation of air quality in general.

New Brunswick: The ambient air quality standards of the province were adopted following a review of studies on the effects of these pollutants and consultation with members of the Federal-Provincial Committee on Air Pollution.

Quebec: Quebec has adopted ambient air quality standards which are similar in general to national air quality objectives with the exception that 24-hour and annual arithmetic means for ozone were not retained. The principal concern of the province is to ensure that a defined ambient air quality standard is designed to protect public health for all segments of the population, and to include adequate safety margins for such protection. Quebec also subscribes to the federal government's idea of protecting the welfare of people i.e. provide adequate protection against adverse effects to personal comfort, well being, animals, vegetation, soil, water, and visibility.

Ontario: The regulation of air emissions in Ontario is achieved by reference to "point of impingement" standards which are design concentrations set to ensure achievement of satisfactory ambient air quality as defined by air quality criteria. These criteria as were established on the basis of adverse effects on human beings, animals, vegetation and property, with the most limiting value usually being chosen. For the pollutants related to automotive emissions, the rationales used are as follows:

Carbon monoxide: human health
Nitrogen dioxide: human health
Ozone: vegetation injury or damage

Manitoba: Manitoba does not maintain ambient air standards but uses the approach of ambient air quality objectives and guidelines. The provincial maximum acceptable and desirable ambient air quality objectives for carbon monoxide, oxidants, and nitrogen were adopted from the federal levels. Provincial ambient air guidelines in the province are based on internal reviews of literature and standards that exist in other jurisdictions. Manitoba has the following non-methane hydrocarbon guidelines:

maximum acceptable level (3-hr average)	160 $\mu g/m^3$ (0.24ppm)
maximum desirable level (3-hr average)	125 $\mu g/m^3$ (0.19ppm)

Even though there is a guideline level for hydrocarbons, it is not used extensively in the province.

Saskatchewan: The province has established ambient air standards based on the Federal desirable objectives. As such, they are probably the most restrictive ambient air standards in effect in Canada. Future changes will probably be in agreement with changes to the National Air Quality Objectives. Saskatchewan Environment may consider recommending a revision to the ozone standard but not before an appraisal of what constitutes natural and anthropogenic levels of oxidants is conducted in the province.

Alberta: Alberta has established maximum level regulations entitled maximum permissible concentrations of air contaminants in the ambient air. Again, Alberta has adopted primarily the National Air Quality Objectives (a mixture of maximum desirable and acceptable levels) for NO_2, CO, and oxidants.

British Columbia: At this time, no levels have been published for NO_2 or oxidants. Ambient air quality criteria are now being developed for approval under the new Environment Management Act.

3.3 Other Countries

3.3.1 United States

The setting of ambient air standards based on scientific criteria was adopted by the United States in their Air Quality Act of 1967 which required that "from time to time, but as soon as practicable, develop and issue to the States such criteria of air quality as in his judgement may be the requisite for the protection of health and welfare. Such criteria shall ... reflect the latest scientific knowledge useful in indicating the kind and extent of all identifiable effects on health and welfare which may be expected from an air pollution agent...". This directive has led to the publication of criteria documents dealing in considerable detail with the effects of specific pollutants. Currently, United States' air standards must be reviewed at least every 5 years to ensure that the standards are based on the latest scientific information.

Ambient air standards are of two types: primary and secondary. A primary standard for a given pollutant is to provide protection of public health, while a secondary standard provides for protection against adverse effects on vegetation, materials, and visibility. For NO_2, O_3, and CO, primary and secondary standards are identical.

The major issues that are addressed in setting National Ambient Air Quality Standards, according to Padgett and Richmond (1983) of the U.S. EPA, are:

(i) allowing for an adequate margin of safety
(ii) dealing with scientific uncertainties

An adequate safety margin entails determining what effects are considered to be adverse and what population groups are particularly susceptible to the effects associated with a given pollutant. The concept of a margin of safety provides for protection against health effects not yet identified or those identified but not well understood.

Currently, the U.S. EPA is attempting to use risk analysis techniques to treat the uncertainties of scientific criteria. Basically, this procedure entails assessing the risks of occurrence of health effects, calculating expected numbers of specified adverse events, and describing the nature and severity of a particular adverse event.

The U.S. EPA produces two major documents in the process of setting standards: a criteria document and a staff paper interpreting studies i the criteria document. A criteria document before being formalized is subjected to public and scientific peer review. The staff paper helps to bridge the gap between science in the criteria document and judgements required in setting ambient standards. This paper also undergoes review by the scientific community and the public.

There is a fundamental difference in approach between that of the Unite States and Canada. The promulgated U.S. standards must be legally defensible in the courts. Therefore the United States tends to rely on selected methodologies out of the full spectrum of criteria. This adversary system can lead to lawsuits against standards which are considered too restrictive or too lenient. For example, the U.S. ozone standard prior to 1978 was 160 $\mu g/m^3$ (80 ppb). As a result of a lawsuit by the petroleum industry, the U.S. EPA was forced to review the standard and subsequently a new standard of 240 $\mu g/m^3$ (120 ppb) was promulgated.

3.3.2 European Countries

In Sweden, the National Environment Protection Board (NEPB) has major responsibility for air pollution. The responsible ministry is agriculture. The NEPB wanted to develop both air quality and source emission standards, but initially only developed emission standards partly due to the inadequate state of scientific knowledge about health and environmental effects. Recently, criteria documents have been prepared for carbon monoxide (Rylander and Vaslerlund, 1981) and nitrogen oxides (G. Perrson, personal communication, 1983) in preparation for establishing ambient air standards for these pollutants.

West Germany has promulgated standards for nitrogen dioxide and carbon monoxide. These standards were first set in 1974 and include provision for long term and short term effects. The long term effect standard is the arithmetic mean of all half hourly averages per year, while the short term effect standard is the 95 percentile of all half hourly averages per year.

Criteria were transmitted to air standards with consideration for the not well defined boundaries between harmful and non-harmful effects. The criteria for each substance are based on the work of the WHO, NATO-CCMS, EEC and the Air Pollution Control Commission of the Association of German Engineers (Weber, 1983). The criteria as a basis for air quality standards are oriented toward preventing:

(i) dangers for health including already affected or sensitive population
(ii) nuisances (e.g. odour)
(iii) disadvantages (e.g. plant, animal injury)

The European Economic Community (EEC) consisting of Belgium, West Germany, Denmark, France, Ireland, Italy, Netherlands, and the United Kingdom has set ambient air quality standards for SO_2 and particulates in 1980, but has not developed standards for ozone, nitrogen oxides or carbon monoxide. Standards are applicable to member countries. The EEC prepared a preliminary air quality criteria document for NO and NO_2 in 1976 (EEC, 1976).

3.3.3 Other Countries

Japan established its first ambient air quality standard (carbon monoxide) in 1970 (IPCA, 1981). Japan first observed photochemical air pollution in 1970 and introduced measures to control photochemical oxidants in 1973 when an air quality standard for oxidants was established. At the same time, a NO_2 standard was also established, mainly because it is one of the precursors of photochemical air pollution (OECD, 1979). The oxidant control strategy for oxidants was originally directed to NO_x control, but hydrocarbon emissions are now receiving attention. The ambient air quality standards in Japan are goals to be achieved and are not legally enforceable. In this respect, Japan's "standards" are similar to Canadian "acceptable air quality objectives" and, to some extent, based on the same considerations. Emission standards are set in order to obtain the goals specified by ambient standards.

Australia is a federation of states and each state has responsibility for implementing air pollution control programmes. In 1981, the State of Victoria published their State Environment Protection Policy (The Air Environment) which provided air quality objectives (called Class indicators) for air pollutants which are widespread in the urban air environment (Victoria Government Gazette, 1981). These concentrations of any Class 1 indicator may not exceed an acceptable level on more than 3 days in any year except for ozone which may not exceed the acceptable level on more than 1 day per year, and must remain below a detrimental level at all times.

The detrimental level refers to a concentration of an indicator at or above which a substantial proportion of the exposed population may be adversely affected or significant changes are likely to be caused to some segments of the environment. The acceptable level refers to a concentration of an indicator at or below which all beneficial uses, listed below, are protected:

(a) life, health and well-being of humans;
(b) life health and well-being of other forms of life, including animals and vegetation;
(c) visibility;
(d) useful life and aesthic appearance of buildings, structures, materials; and
(e) aesthetic enjoyment and local amenity

Design ground level concentrations, similar to the Province of Ontario's point of impingement standards, were set to attain the air quality objectives.

Australia has a photochemical pollution problem in its large urban centres; the State of Victoria'a objectives include provisions for protection of visibility and of vegetation.

New Zealand uses the WHO recommended levels as guidelines (OECD, 1979). Some concern has been expressed that photochemical pollution may develop (OECD, 1981).

3.4 Previous, Present, and Proposed Air Quality Standards, Objectives, and Guidelines

This section explores the rationale used in establishing standards or guidelines by the World Health Organization, Canada, and the United States. Standards or objectives are also presented from West Germany, Japan, and the State of Victoria in Australia for comparison only. The scientific criteria are presented in detail in Chapter 4 of this report.

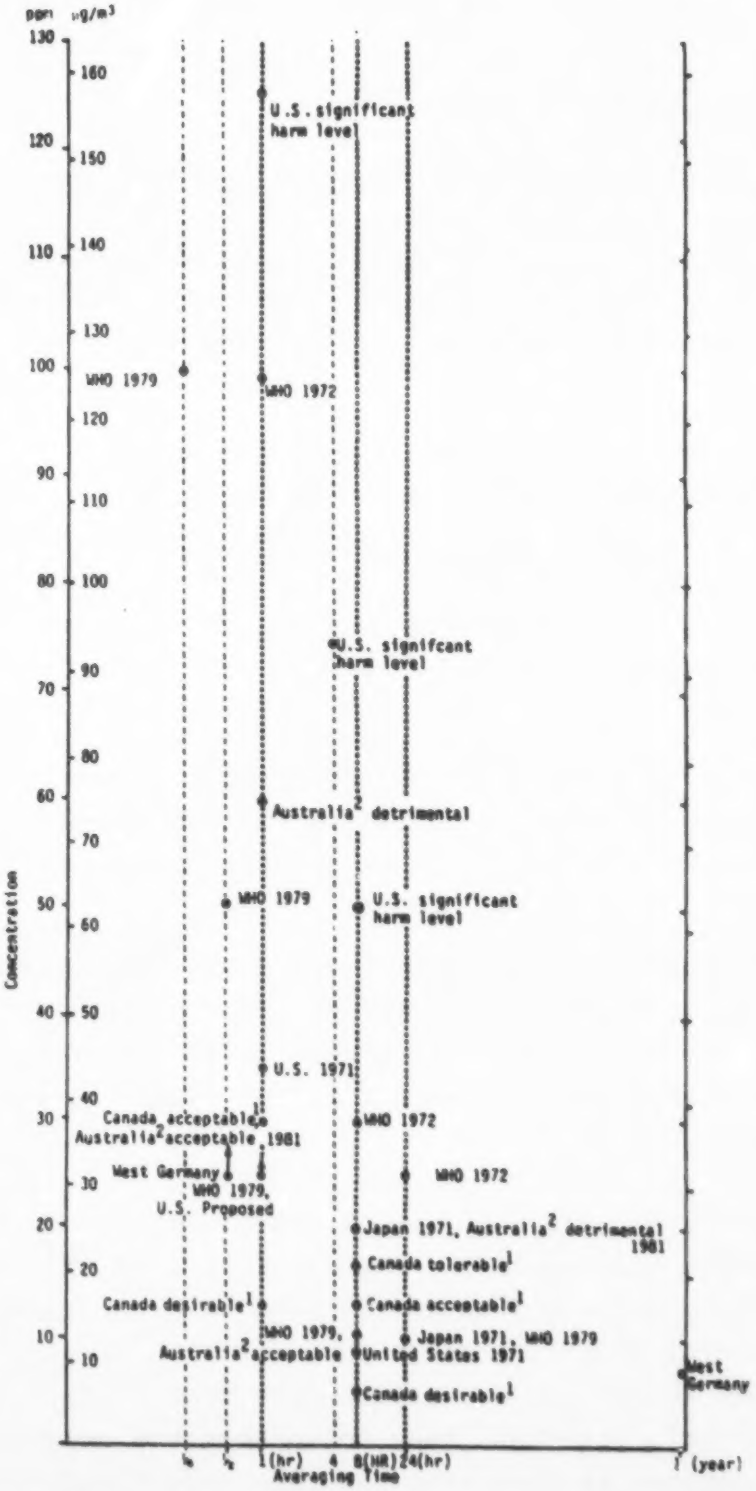
3.4.1 Carbon Monoxide

In determining exposure levels for carbon monoxide, concern is principally with public health effects. Carbon monoxide is rather unique among the constituent pollutants of automobile emissions in that it has a biological indicator of potential effects. CO combines with haemoglobin to form carboxyhaemoglobin (COHb) which results in the displacement of oxygen and interference with oxygen delivery to tissues. Uptake of CO by blood increases with increasing CO concentrations in air, increasing length of exposure, and increasing breathing rates. Carbon monoxide is also produced endogenously in man resulting in a normal background level of about 0.5% COHb in blood.

The critical concentration of CO in health effects studies refer to carboxyhaemoglobin (COHb) in blood. This has to be correlated with CO concentration in the ambient atmosphere. Thus, there are usually two safety margins built into CO standards, one for COHb levels and the second for correlation of ambient CO and COHb.

Existing, previous, and proposed ambient air quality standards for CO are provided in Figure 3.4-1. All of the "acceptable" standards, objectives, and guideline CO levels lie between the Canadian maximum desirable levels and the United States significant harm levels. The scatter of levels of CO air quality standards between the above mentioned two extremes reflects the different interpretations, assumptions, and, to some extent, better recent knowledge on adverse health effects.

FIGURE 3.4-1
CARBON MONOXIDE STANDARDS, OBJECTIVES, AND GUIDES



1. Canadian tolerable, acceptable, and desirable values refer to maximum levels

2. Objectives listed are for the State of Victoria, Australia.

The following discusses Canadian, WHO, and United States rationales for recommending or setting the levels indicated in Figure 3.4-1.

Canadian Air Quality Objectives:

The CO national objectives levels recommend in 1971 were:

Maximum desirable limits:	1-hr. average 15 mg/m^3	(13 ppm)
	8-hr. average 6 mg/m^3	(5 ppm)
Maximum acceptable limits:	1-hr. average 35 mg/m^3	(30 ppm)
	8-hr. average 15 mg/m^3	(13 ppm)

Subsequently, a maximum tolerable level of 20 mg/m^3 (17 ppm) for an 8-hour average was established. The specific rationale for establishing the tolerable level was never published (the general definition of this level is specified in Table 3.2-1), so the subsequent discussion deals only with desirable and acceptable limits. The Subcommittee on Air Quali Objectives considered the following guidelines (Sub. on Air Quality Objectives, 1976):

(i) in order to protect all sensitive groups of the general population, ambient air concentrations of carbon monoxide should be such that COHb levels do not exceed 5% saturation in non-smokers.

(ii) in view of repeated suggestions of a significant effect on performance at COHb levels between 2.5 and 5%, it is reasonable to apply a safety factor or margin to the definite effect level of 5% COHb and aim at COHb saturation levels below 3% in non-smokers.

The Subcommittee then utilized 4 to 5% COHb levels to prescribe the maximum acceptable limits and the 2 to 3% COHb level for the maximum desirable limits. At more than the 5% COHb level, they noted evidence existed for physiological stress on patients with heart disease; impairment in performance of some psychomotor tests; and impairment of visual acuity. At 3.95% COHb, they noted that the oxygen debt increases. Suggestions at that time had been made about time discrimination below 3% COHb, and no detrimental effects had been noted below 2% COHb.

To relate CO ambient concentrations to acceptable COHb levels, the committee utilized an equation developed by Laksek and Burke (1969), assuming that moderate to strenuous exercise was possible over a 1-hour period and resting to moderate activity was representative of conditions during an 8-hour period. However, they did not specifically say what parameter values they used in determining this relationship. The committee is now in the process of reviewing the CO criteria and is utilizing the more commonly accepted Coburn model to relate CO to COHb levels (H. P. Sanderson, personal communication, 1983).

World Health Organization Guides: In 1972, the report of the WHO expert committee agreed that individuals should be protected against continuous COHb levels of approximately 4% or over (WHO, 1972). In their evaluation of the recommended guidelines, they considered that smokers may exceed 4% COHb and that since equilibration at 4% could be produced by constant inhalation at 29 mg/m^3 (25 ppm, 24-hour average), this concentration was undesirable. The shorter term levels of ambient CO were determined by the time required to reach 4% COHb and were set at 35 mg/m^3 (30 ppm) over 8-hour and 117 mg/m^3 (100 ppm) 1 hour. At this time, they debated whether susceptible persons or smokers were the proper subjects for protection. Their basis for the 4% COHb level was that above this concentration, there appeared to be an increased risk for persons with cardiovascular disease.

The WHO in 1979 revised their original recommended COHb level and arrived at a tentative recommendation by considering new evidence on the exposure limit for persons with cardiovascular illness to carbon monoxide in conjunction with exercise (WHO, 1979a). A range of carboxyhaemoglobin concentrations of 2.5-3.0% was recommended as agreement was not reached on a single level. The WHO task group recommended ambient air CO guidelines that would prevent COHb levels exceeding 2.5-3% in general non-smoking populations using the results of Coburn's model and comparison with other models that relate ambient CO exposure to blood COHb. These levels are presented in Table 3.4-1.

TABLE 3.4-1

Guidelines for exposure conditions to prevent carboxyhaemoglobin levels exceeding 2.5–3% in nonsmoking populations

---

(*a*) A ceiling or maximum permitted exposure of 115 mg/m^3 (100 ppm) for periods of exposure not exceeding 15 min (No exposure over 115 mg/m^3 (100 ppm) permitted, even for very short time periods).

(*b*) A time-weighted average exposure of 55 mg/m^3 (50 ppm) for periods of exposure not exceeding 30 min.

(*c*) A time-weighted average exposure of 29 mg/m^3 (25 ppm) for periods of exposure not exceeding one h.

(*d*) A time-weighted average exposure of 15 mg/m^3 (13 ppm) for periods of exposure of more than one h.

(*e*) A time-weighted average exposure of 11.5 mg/m^3 (10 ppm) for periods of exposure of 8–24 h.[a]

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[a] Suggested by the Secretariat.

The WHO task group commented that further development of Coburn's model concepts will improve the basis on which theoretical uptakes can be calculated.

United States Standards:

In 1971, both the primary and secondary standards were set at levels of 10 mg/m^3 (9 ppm), 8-hour average and 40 mg/m^3 (35 ppm), 1-hour average, neither to be exceeded more than once per year. The national primary standard was based on evidence that low levels of carboxy-haemoglobin in human blood may be associated with impairment of ability to discriminate time intervals. This standard incorporated protection of the health of persons, including sensitive groups, to the effects of COHb levels above 2%, which was believed to provide an adequate safety margin and protect against known and anticipated effects.

In 1980, the U.S. EPA proposed to retain the existing 8-hour primary standard at 10 mg/m^3 (9 ppm) and to lower the primary 1-hour standard to 29 mg/m^3 (25 ppm) (Federal Register, 1980). The change in the 1-hour standard was proposed because of EPA's recognition that more rapid accumulation of COHb occurred in moderately exercising sensitive persons, compared with individuals at rest. In the 1971 standard, the impact of excercise, which is greater for short duration exposure, was not considered in the original standard. The U.S. EPA also noted that a secondary standard was not appropriate because environmental effects have only been observed at very high levels. Also proposed was changing to a statistical standard in place of the deterministic standard in place. A certain number of exceedances would be stated as an expected value, not as an explicit value (i.e. 1 exceedance per year in 1971 standard). The EPA proposed that exceedances would be determined on the basis of number of days in which 1-hour and 8-hour concentrations are above standard levels.

The factors considered in selecting a margin of safety and standard level were (EPA, 1979):

(i) the 1978 Aronow Study which indicates that adverse effects in angina patients are associated with COHb levels in the range of 2.5-3.0%.

(ii) evidence in animal studies that the developing fetus is exposed to higher COHb concentrations than the mother for long-term CO exposures.

(iii) a margin of safety is required to account for the uncertainties in the relationship between ambient CO exposure and resulting COHb levels.

(iv) a margin of safety is required to protect against adverse effects of sensitive visitors to high altitude areas, who are not adapted to these high altitude sections.

(v) the increased risk for anaemics and other individuals, whose uptake of CO is greater, should be considered in determining an adequate margin of safety.

(vi) little or no evidence exists for the need of a more restrictive national ambient air quality standard to protect smokers from a possible incremental COHb burden from the air.

(vii) the bolus effect (the uncertainty relating to adverse health effects from short duration, 5-10 minutes, high-level exposure) is of concern in selecting a margin of safety, but does not appear to be an overriding consideration in the determination of a standard level.

In their analysis of CO uptake uncertainty, the EPA stated that only a very small fraction of sensitive groups (cardiovascular and peripheral vascular disease) would reach a final COHb level of 3.0% for a 29 mg/m^3 (25 ppm) standard. In the Federal Register notice, the EPA also indicated that the COHb levels associated with 10 mg/m^3 (9 ppm) 8-hour and 29 mg/m^3 (25 ppm) 1-hour standards do not afford a large degree of protection for sensitive persons and subsequently they invited discussion on whether an adequate margin of safety was provided by the factors they had considered.

The significant harm levels for CO (exposure levels that constitute an imminent and substantial endangerment to the health of persons) established in 1971 were associated with a 5 to 10% COHb concentration as the critical range to be avoided. These levels and averaging times are:

57 mg/m^3	(50 ppm)	8-hour average
86 mg/m^3	(75 ppm)	4-hour average
109 mg/m^3	(125 ppm)	1-hour average

The 1980 EPA notice proposed no modifications to these 1971 standards for significant harm.

Summary: The review of past, existing, and proposed standards for carbon monoxide suggests a declining COHb level which is used to delineate adverse health effects. There is also more recognition of the rapid accumulation of COHb by exercising individuals. Canada's current CO maximum acceptable limit of 35 mg/m^3 (30 ppm) for 1-hour, recommended in 1971, is 6 mg/m^3 (5 ppm) higher than the more recent WHO recommendations and the proposed United States standards. Similarly, the Canadian acceptable limit for 8-hour exposure is higher than the existing United States standard and the 1979 WHO recommendation. Only the Japanese 8-hour standard which was set in 1971 is higher than its Canadian counterpart.

All of the CO standards set or recommended are directed at the protection of vulnerable groups such as cardiac patients and anaemics from adverse effects. As indicated in the United States approach, there is the concept of providing a margin of safety for sensitive groups in visiting high altitude locations (e.g. a cardiac patient visitor from Halifax in Calgary), the fetus, and the uncertainty in relationships between CO exposure and COHb blood levels.

The Coburn model was used by the U.S. EPA in proposing their ambient air standard (EPA, 1979) and heavily relied on by the WHO. Currently, one of the aspects being explored by the U.S. EPA and the Canadian Subcommittee is the sensitivity of the Coburn model predictions of COHb levels associated with variable CO exposures. For example, Biller and Richmond (1982) have extended the static Coburn analysis to a dynamic situation using actual monitored values of CO. Their estimates indicate that the assumption of constant CO concentration for an 8-hour averaging time can lead to underestimating the COHb response to CO concentrations that are actually fluctuating on a much shorter time scale. Further elaboration of this aspect is provided in the CO health effects section of Chapter 4.

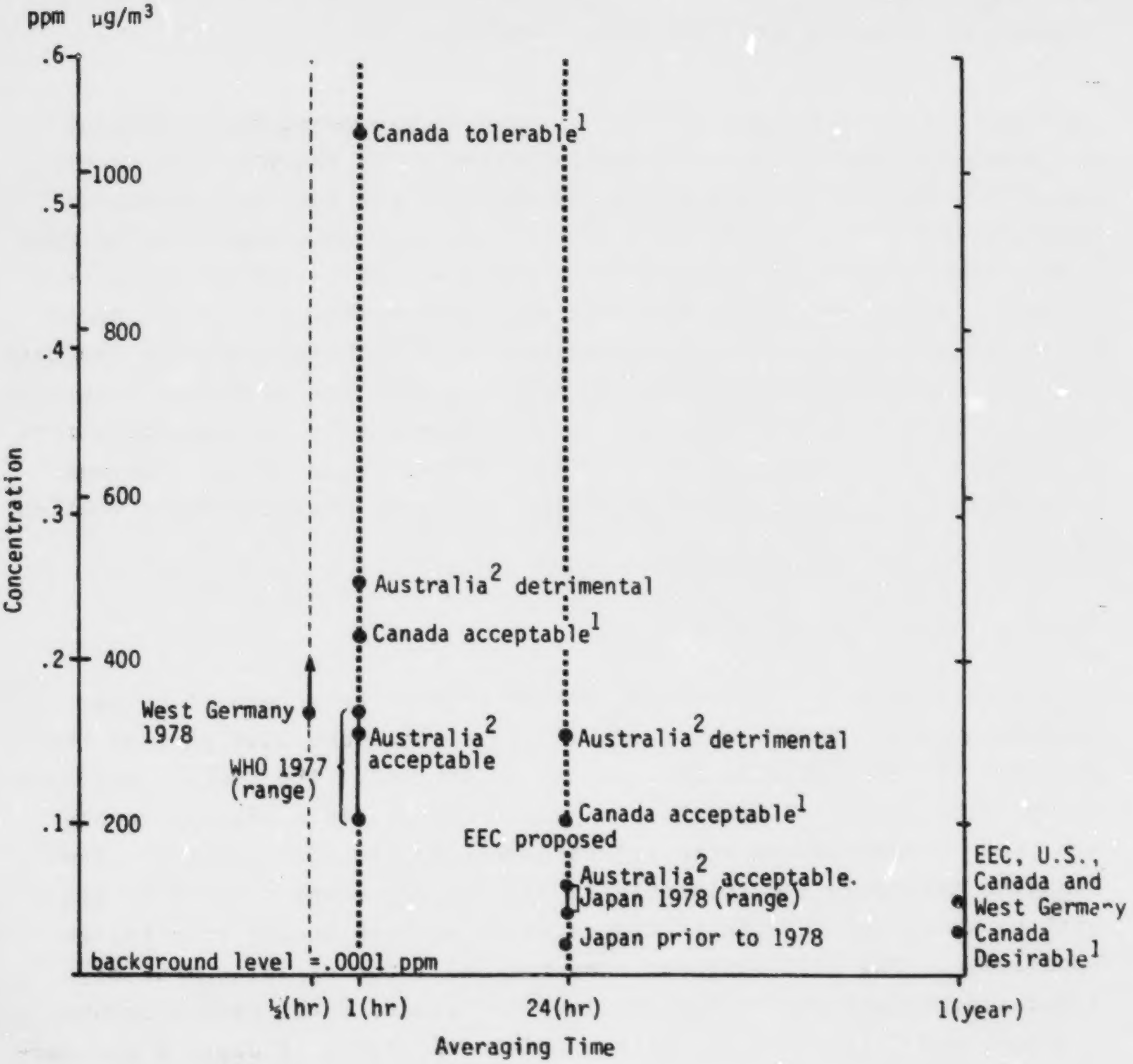
3.4.2 Nitrogen Oxides

In the setting of NO_2 standards, health effects have been of primary consideration. Consideration of welfare effects are also part of the criteria for the Canadian air quality objectives. The lowest standards shown in Figure 3.4-2 are those of Japan; permissible oxidant levels are used in combination with standards for NO_2 in this country. The Canadian desirable and tolerable levels are representative of levels with no known health effects and definite adverse health effects, respectively. The following explores the basis for establishment of standards, objectives, and guidelines by Canada, World Health Organization, and United States, with a brief discussion of Japan's and Germany's standards.

Canada: The National Air Quality Objectives for NO_2 are based in part on nuisance effects observed as odour perception, as well as epidemiological studies such as the Chattanooga study. (Shy et al. 1970a, b).

FIGURE 3.4-2

NITROGEN DIOXIDE STANDARDS, OBJECTIVES, AND GUIDES



1. Canadian tolerable, acceptable, and desirable values refer to maximum levels

2. Objectives listed are for the State of Victoria, Australia.

of extreme controversy due to analytical errors and methodological criticisms. These aspects are discussed further in the NO_2 health effects section of Chapter 4. Consideration was also given to synergistic effects with photochemical oxidants and SO_2, and to the possibility of lower than actual values being monitored.

The maximum acceptable limits were based on the following criteria:

1-hour 400 $\mu g/m^3$ (.21 ppm)	a) the level is slightly below odour perception by the majority of young, healthy people at 410 $\mu g/m^3$ (0.22 ppm) b) provides a safety margin of 10 for protection against air flow resistance in the presence of equal concentrations of SO_2 c) lowest concentration shown to be harmful to animals is 433 $\mu g/m^3$ (0.23 ppm)
24-hour 200 $\mu g/m^3$ (.10 ppm)	- no evidence of health effects at this level even with a concentration of 260 $\mu g/m^3$ (0.10 ppm) SO_2
1-year 100 $\mu g/m^3$	- based on the interpretation of the Chattanooga investigations prevalent at the time i.e. slightly below the levels shown to have adverse effects; these studies suggested respiratory effects in children

The criteria document also notes that plant damage occurs as a result of synergistic effects on exposure to a mixture of 200 $\mu g/m^3$ (0.10 ppm) NO_2 and SO_2 over 4-hours which presumably was used as a criteria for the 1- and 24-hour levels. The maximum desirable limit of 60 $\mu g/m^3$ (0.03 ppm) NO_2 annual average was recommended based on the evidence that there were no known acute human health effects; no abnormal effects observed on materials; and that animals and vegetation are generally more resistant than humans. No documentation on the rationale for the maximum tolerable level is available at this time.

In a recent review of NO_2 health effects prepared by B.G. Ferris Jr. for Health and Welfare Canada (EHD, 1982) as part of the current review of NO_2 ambient air quality objectives, the following levels were recommended for NO_2 maximum acceptable air quality objectives:

1-hour	750 $\mu g/m^3$	(0.4 ppm)
24-hour	470 $\mu g/m^3$	(0.25 ppm)
Annual	100 $\mu g/m^3$	(0.05 ppm)

These levels have been endorsed by Health and Welfare Canada but do not include consideration of welfare effects, such as affects on vegetation and ecosystem, materials, vegetation etc.

The 1-hour 750 $\mu g/m^3$ (0.4 ppm) was selected because it appeared to provid a no-effect level with some safety margin based on observed effects of NO_2 on the lung function of asthmatics at 940 $\mu g/m^3$ (0.5 ppm), and developmen of only mild symptoms by asthmatics at 560 $\mu g/m^3$ (0.3 ppm). There were no effects in healthy subjects following exposure to 2800 $\mu g/m^3$ (1.5 ppm) for one to two hours.

A recommended 24-hour objective was based on experimental evidence for shorter durations of exposure:

(i) no effects from 756 $\mu g/m^3$ (0.42 ppm) NO_2 in conjunction with other pollutants
(ii) effects on healthy office workers at 1504 $\mu g/m^3$ (0.8 ppm) NO_2
(iii) no effects from daily concentrations of 361 $\mu g/m^3$ (0.19 ppm) if 1 to 2-hour samples are representative of this time period

As is the case with the U.S EPA review of their NO_2 standard, Ferris points out that the Chattanooga epidemiological study no longer supports an annual average of 100 $\mu g/m^3$ (0.05 ppm). However, based on other epidemiological evidence, it was recommended that annual concentration of 100 $\mu g/m^3$ (0.05 ppm) would carry minimal risk and be an acceptable level even for sensitive populations.

The World Health Organization: The WHO air quality criteria and guide report of 1972 concluded that insufficient information was available on the effects of nitrogen oxides on humans at that time upon which to base air quality guides (WHO, 1972). In 1977, the WHO committee was still faced with inconclusive epidemiological evidence on human health, but felt it was appropriate and prudent to use available controlle study data on animals and humans to recommend air quality guides for NO_2 (WHO, 1977). Based on these data, the WHO committee selected a NO_2

level of 940 $\mu g/m^3$ (0.5 ppm) as an estimate of the lowest observed effect-level for short-term exposures. The adverse effect relates to increased airway resistance of lungs which could play a role in causing respiratory disease. They also noted that one controlled human study on a sensitive group, asthmatics, showed an adverse effect at a lower concentration of 190 $\mu g/m^3$ (0.1 ppm) NO_2. Because of the uncertainty about the lowest adverse effect level for sensitive groups, they concluded a safety margin was required; as NO_2 has a high biological activity, the task group believed that the margin of safety should be considerable. Based on consideration of existing NO_2 levels in large cities, which are in the vicinity of the known adverse effect of 940 $\mu g/m^3$ (0.5 ppm), the Task Group proposed a minimum safety factor of 3 to 5, giving a recommended guide level of 190 to 320 $\mu g/m^3$ (0.10 to 0.17 ppm) for a 1-hour exposure. Provision for one exceedance per year was attached to this recommended level. The Task Group found insufficient information upon which to recommend a long-term exposure level for NO_2 and no evidence that NO concentrations in the ambient air have a significant biological effect.

The existing United States annual standard of 100 $\mu g/m^3$ (0.053 ppm) was based largely on the Chattanooga community epidemiology study. The recent EPA review of NO_2 standards (EPA, 1982) concludes that this study is no longer seen as an adequate basis for retaining the existing standard and that other outdoor epidemiological studies are either flawed or report no effects associated with NO_2 exposure. The review recognized that there was evidence that short-term peaks of NO_2 might cause adverse health effects in children. Studies in homes with gas stoves suggest that multiple exposures to NO_2 levels above 940 $\mu g/m^3$ (0.5 ppm) should be avoided and that repeated peaks in the range of 280 $\mu g/m^3$ to 560 $\mu g/m^3$ (0.15 to 0.30 ppm) may be of concern.

Two approaches were suggested by the U.S EPA to minimize health effects. The first was to retain an annual standard between 100 $\mu g/m^3$ and 150 $\mu g/m^3$ (0.05 and 0.08 ppm) to provide protection against short-term peaks. Based on existing air quality data in the United States, a 150 $\mu g/m^3$ (0.08 ppm) standard would be expected to limit the number of days with 1-hour peak concentrations above 368 $\mu g/m^3$ (0.30 ppm) to about ten per year. For a 100 $\mu g/m^3$ (0.05 ppm) standard, the hourly peak concentration exceeding

10 to 20 days per year would be 184 $\mu g/m^3$ (0.15 ppm). An annual standard in this range would also preclude a 1-hour peak concentration of 940 μg/m (0.5 ppm). The alternative approach, of course, was to establish a short-term standard somewhere below 940 $\mu g/m^3$ (0.5 ppm) rather than using a annual standard as a surrogate indicator. The final recommendation was to provide for an annual standard in the lower range of 100 to 150 $\mu g/m^3$ (0.05 to 0.08 ppm) based on the practical advantage of not requiring formulation and implementation of a new regulatory program based on 1-hour standards.

The EPA staff paper also concluded that there was no need to provide a separate secondary standard if the primary standard was within their suggested range to protect human health. The welfare aspects considered in the review included personal comfort and well-being (symptomatic effect causing personal discomfort e.g. headaches, dizziness), vegetation effects, visibility impairment, and material damage. These effects are either not quantifiable due to lack of information, or protection is afforded by the primary standard.

Japan first observed photochemical air pollution effects in 1970 when students in the outskirts of Tokyo were apparently injured (cited by Nakamo, 1980). In 1973, an air quality standard of 40 $\mu g/m^3$ (0.02 ppm) (daily average) was established because NO_2 has adverse effects on health and is one of the precursors of photochemical air pollution. In 1978, the standard was revised upward to a daily average in the range of 80-120 $\mu g/m^3$ (0.04 - 0.06 ppm).

West Germany released standards for NO_2 and NO in 1974 on the basis of health effects and the role of NO_x as precursors of photochemical air pollution (EEC, 1976). The standards set are:

NO_2	NO	
0.05 ppm(100 $\mu g/m^3$)	0.17 ppm(200 $\mu g/m^3$)	arithmetic mean annual value based on ½-hour mean values
0.16 ppm(300 $\mu g/m^3$)	0.50 ppm(600 $\mu g/m^3$)	95% value of the cumulative frequency distribution based on ½-hour mean values

The sole purpose of the NO ambient standard is prevention of the formatio of photochemical air pollution and NO_2; no health effects are associated with such low levels of NO.

The EEC proposed values of NO_2 and NO for the protection of the health as follows (OECD, 1979):

NO	308 $\mu g/m^3$	(0.25 ppm)	in winter maximum daily mean
	185 $\mu g/m^3$	(0.15 ppm)	in summer maximum daily mean
NO_2	188 $\mu g/m^3$	(0.10 ppm)	in maximum daily value
	94 $\mu g/m^3$	(0.05 ppm)	annual mean

The criteria were based on direct health effects of NO_2 and were independent of any role NO_x might play in photochemical air pollution.

The long-term standards for NO_2 for all countries are in the vicinity of 100 $\mu g/m^3$ (0.05 ppm) NO_2 based on the protection of health for sensitive groups of the population. Of the countries with short-term standards (daily or 1-hour averages), the prime criteria has been health effects, but consideration has also been given to the role of NO_2 in formation of photochemical pollution (Japan and West Germany), plant damage (Canada), and sensory perception (Canada). The scientific criteria available for a long-term standard based on health effects is very tenuous and the WHO as of 1977 found insufficient information to recommend an air quality guide. The U.S. EPA review of scientific criteria also reached this conclusion, but since an annual standard was in place, it was felt that this level could be used as a surrogate for protection against short-term peaks of NO_2. An appreciable safety factor of 3 to 5 was incorporated into the 1-hour WHO health guideline because or the high biological activity of the NO_2, while a recent recommendation to Health and Welfare Canada appears to have safety factor of about 0.2.

3.4.3 Hydrocarbons

There are no air quality objectives for hydrocarbons in Canada. In 1983, the United States revoked its non-methane hydrocarbons standard of 160 $\mu g/m^3$ (0.24 ppm) 3-hour average for the period from 6 to 9 a.m.

(Federal Register, 1983). The past standard had been unique in that the levels set were not based on direct health or welfare effects of hydrocarbons, but was intended to be used as a guide in helping determine the emission reductions necessary for attaining the photochemical oxidant standard. The standard was revoked because no consistent quantitative relationship exists in the United States between ambient air zone levels and hydrocarbon levels. The U.S. EPA also noted that their review of adverse health and welfare effects indicated no basis for retaining the standard.

In August 1976, the Central Council for the Control of Environmental Pollution in Japan set a guideline of 133 to 207 mg/m^3 (0.2 to 0.31 ppm) for non-methane hydrocarbon concentrations during 6-9 a.m. (OECD, 1979). This was set to achieve the ambient air quality standard for oxidants. The province of Manitoba has similar guidelines (cf. Section 3.2.2).

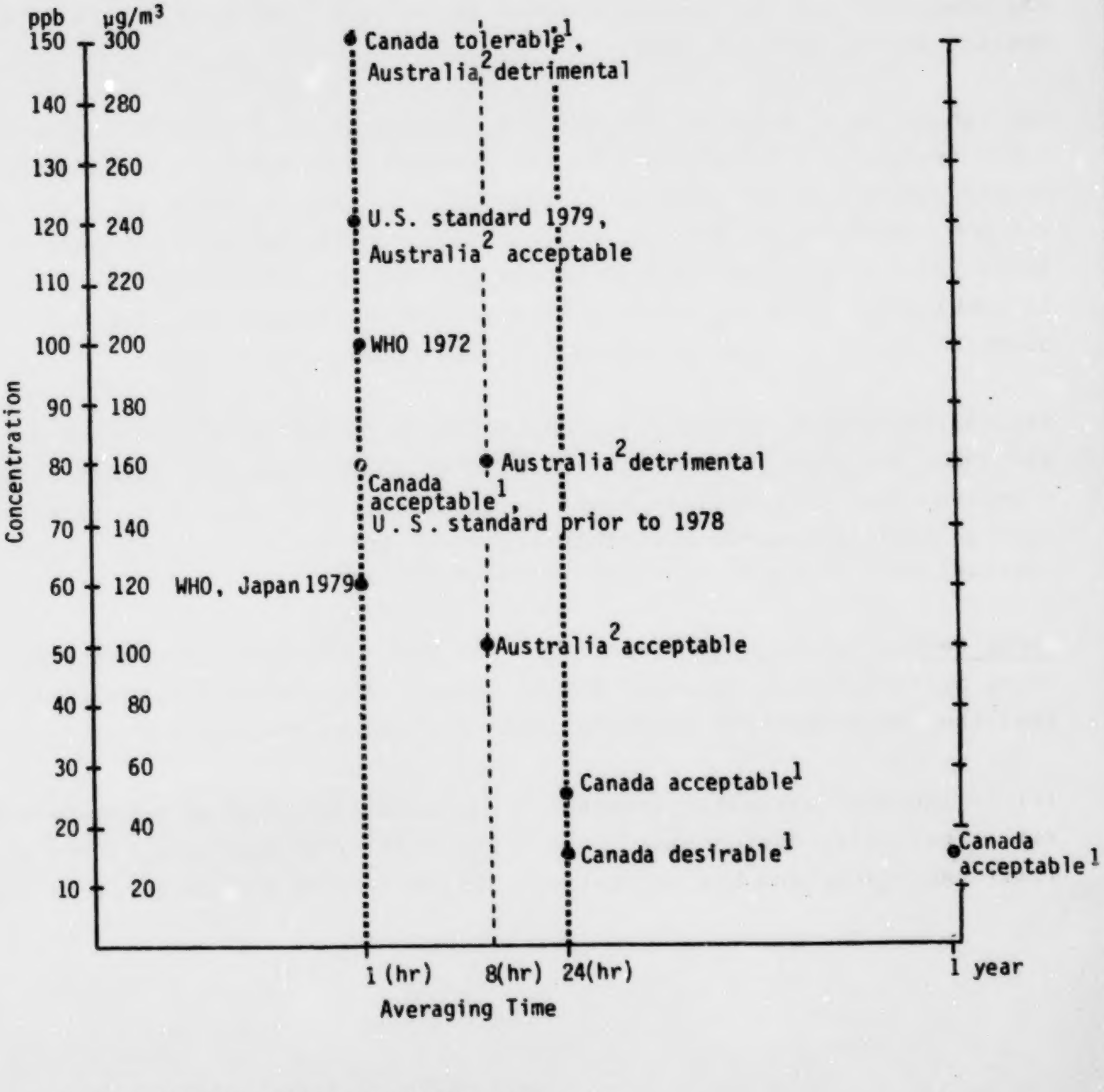
3.4.4 Photochemical Oxidants

Standards, objectives, and guidelines for ozone from various countries are presented in Figure 3.4-3. In establishing these standards, the majority of levels set in the early 1970's refer to total oxidant concentrations. More than 90% of the total oxidants are usually in the form of ozone (WHO, 1979). Thus, the results of ambient air monitoring are normally expressed as concentrations of ozone. In more recent years, since monitors became available for monitoring ozone, revised standards have been set for ozone only.

Canada: The oxidant objectives in Canada are based on ozone, not total oxidants. The criteria utilized to recommend limits for air quality objectives included health effects, plant damage, material degradation,

FIGURE 3.4-3

OXIDANT AND OZONE STANDARDS, OBJECTIVES, AND GUIDES



1. Canadian tolerable, acceptable, and desirable values refer to maximum levels

2. Objectives listed are for the State of Victoria, Australia.

and natural background levels. The maximum acceptable limit of 160 $\mu g/m^3$ (80 ppb) for a 1-hour average was recommended on the basis that this concentration was above the minimum level for impairment of performance of student athletes at 140 $\mu g/m^3$ (70 ppb) and at the level of experimental animal evidence, indicating increased susceptibility of mice to bacterial infection at 160 $\mu g/m^3$ (80 ppb) ozone for 30-hours. No specific rationale was presented for the 24-hour average of 50 $\mu g/m^3$ (25 ppb) or the 1-year average of 30 $\mu g/m^3$ (15 ppb).

Desirable limit criteria included the evaluation that no known acute or subacute health effects occur at the recommended levels. The Subcommitte on Air Quality (1976) also noted that natural rubber cracks at a 40 $\mu g/m^3$ (20 ppb) exposure for 1-hour; slight injury occurs to peanut plants at 40-60 $\mu g/m^3$ (20-30 ppb) for 24-hours; and synergistic effects of ozone in combination with SO_2 cause damage to tobacco plants at levels of about 60 $\mu g/m^3$ (30 ppb) ozone over 2 to 4 hours.

Natural background levels were considered to be 120 $\mu g/m^3$ (60 ppb) maximu and under 100 $\mu g/m^3$ (50 ppb) 99.5% of the time. Thus, the committee recognized that difficulties might be encountered at certain times of the year in meeting maximum desirable limits of 100 $\mu g/m^3$ (50 ppb) (1-hour average), and 30 $\mu g/m^3$ (15 ppb) (24-hour average).

World Health Organization: In 1972, the WHO (WHO, 1972) recommended total oxidant levels associated with various effects of human beings, that they be considered as appropriate for use as air quality guides:

(i)	increase asthmatic attacks	250 $\mu g/m^3$ (125 ppb as O_3)	1-hr average
(ii)	pulmonary dysfunction	200 $\mu g/m^3$ (100 ppb as O_3)	1-hr averag
(iii)	annoyance and eye irritation	200 $\mu g/m^3$ (100 ppb as O_3)	1-hr averag

The criteria document of 1978 (WHO, 1978) noted that evidence from one controlled human exposure study indicated that exposure to an ozone level of 200 μg/m^3 (100 ppb) for 2-hours can cause some lung obstruction in healthy human subjects. Based on this study and others, the WHO concluded that the first adverse effects can be expected to occur at ozone levels exceeding 200-500 μg/m^3 (100-250 ppb). They noted that animal studies support these results and some effects have been observed at levels below 200 μg/m^3 (100 ppb). The WHO consequently recommended a 1-hour exposure of 100 to 200 μg/m^3 (50 to 100 ppb) with a best estimate of 120 μg/m^3 (60 ppb) to be used as a guideline for the protection of public health. They indicated that a substantial safety factor could not be applied because of natural levels of ozone reaching 120 μg/m^3 (60 ppb), and the difficulty of attaining lower levels, especially in urban areas. The setting of the lower limit of the recommended range below natural levels recognizes that there may be no threshold level for ozone (OECD, 1979). Japan has set a total oxidant standard of 120 μg/m^3 (60 ppb) within the recommended range of the WHO (Japan Environment Agency, 1976).

United States: Prior to 1979, the United States standard was 160 μg/m^3 (80 ppb), 1-hour average, for total oxidants. Under pressure from the petroleum industry (Marshal, 1978) the standard was revised in 1979. The definition of the standards was narrowed only to ozone, and the primary and secondary standards were set at a level of 240 μg/m^3 (120 ppb), 1-hour average. The definition of standard attainment was defined as when the expected number of days per calendar year with maximum hourly average concentrations of 240 μg/m^3 (120 ppb) are equal to or less than one.

The key areas considered by EPA in setting the ozone standard were (Federal Register, 1979):

1. Threshold concept - the adverse health effect threshold concentration for O_3 is unknown, necessitating a margin of safety.
2. Ozone health effects - effects data attributed pulmonary irritation to ozone at short-term ozone concentrations between 300 and 500 μg/m^3 (150 and 250 ppb).

3. **Effects on asthmatics** - based on re-evaluation of evidence for the previous standard, it was considered that a peak hourly oxidant concentration of 500 μg/m^3 (250 ppb) caused elevated asthmatic attacks.

4. Toxicologic findings - there is increased susceptibility to bacterial infection in laboratory animals exposed to 200 μg/m^3 (100 ppb) ozone.

5. Pollutant interaction - SO_2-O_3 synergistic findings support the need for a margin of safety.

6. Welfare effects - materials and vegetation effects were noted.

The EPA also concluded that there was no quantitative evidence to consider potential ozone effects on the aging process and mortality. The EPA decided not to set a secondary standard for protection against damage to plants based on the consideration that an adverse effect must be based on observed growth and yield reductions in field conditions. These data indicate that growth and yield response are related to growing season means of daily maximum 6 and 8-hour-average O_3 concentrations. With attainment of the primary standard, it was concluded that there was no evidence to suggest a significant decrease in growth and yield.

Summary: The chronological history of setting of standards and recommendation of guidelines indicates conflicting trends. The United States relaxed their standard from 160 μg/m^3 (80 ppb) to 240 μg/m^3 (120 ppb) in 1979, while the WHO in the same year reduced their guidelines for protection of human health from the range of 200-250 μg/m^3 (100-125 ppb) to 100-200 μg/m^3 (50-100 ppb). These differing interpretations are essentially based on the same set of scientific criteria, although the WHO tends to use animal data more than the United States in support of their recommendations.

Overall, ozone standards have been set primarily to protect public health. In Canada, vegetation damage was also considered in establishing maximum acceptable objectives. The province of Ontario's air quality criteria for ozone is primarily based on avoidance of vegetation damage (cf. Section 3.2.2) where crop damage attributed to photochemical oxidants has been documented.

3.4.5 Diesel Exhaust Particulates

Particulate standards set in various countries refer to total suspended particulates, smoke measurements, and visibility reduction. The term total suspended particulates usually refers to particles having a size range of 0.001 to 100 μm. A division of this range is usually made at 2 μm with particles below 2 μm called fine and those above called coarse. Diesel exhaust particulates are found almost exclusively in the fine mode. Since particle mode is usually different chemically and has different origins and fates in the atmosphere, there is no quantitative relationship between fine particles and total suspended particulates.

Canadian National Air Quality Objectives do exist for total suspended particulates. One of the recommendations of the Panel on Aerosols of the National Research Council of Canada (NRCC, 1982) was that "The Canadian air quality objective for suspended particulates, that is based on visibility impairment as a criterion, be modified to reflect the relationship between visibility and atmospheric fine particle concentration." Visibility reduction can be correlated with the atmospheric mass loading of fine particles, depending on certain conditions e.g. geographical, location, relative humidity and type of aerosol (ibid, 1982). The Australian State of Victoria has established an acceptable visibility level of 20 km over a 1-hour period for visibility reducing particulates based on aesthetic considerations (Victoria Government Gazette, 1981). The visibility is derived from measurements with an integrating nephelometer, which measures atmospheric light scatter, at relative humidities less than 70 percent. (Section 4 explains, in more detail, the concepts of visibility measurement). This air quality objective is primarily influenced by the atmospheric mass loadings of fine particles.

The OECD method of monitoring referred to as smoke measurement restricts samples to respirable range (approximately less than 10 to 15 μm in diameter) which are more closely related in size to diesel particles. Based on this monitoring methodology, the WHO (1979b) recommended exposure limits in the presence of SO_2 consistent with protection of public health:

	Concentration ($\mu g/m^3$)	
	SO_2	Smoke
24-hour mean	100 - 150	100 - 150
Annual arithmetic mean	40 - 60	40 - 60

Recently, the United States has investigated the possibility of establishing an inhalable particulate standard (EPA, 1982). In addition, the Canadian Subcommittee on Desirable and Acceptable Air Quality Objectives is also in the process of considering whether existing evidence supports the recommendation of an air quality objective for inhalable particulates

The major concern for diesel particulates relates to their potential carcinogenicity (see Section 4). These particles are known to contain cancer-producing agents such as benzo(a) pyrene.

4. EFFECTS OF AUTOMOTIVE AIR POLLUTANTS

4.1 General Introduction

The interaction of a receptor and an air pollutant may result in some kind of effect. The nature, scale, and significance to man of this effect is the heart of the value judgment used in setting standards. The purpose of this chapter is to provide air quality criteria for CO, NO_2, O_3, hydrocarbons, diesel exhaust particulates, and acid precipitation. Whenever possible, dose-response information is provided in the five areas of human concern:

- health
- terrestrial systems (e.g. crops, vegetation)
- aquatic systems (e.g. surface water quality)
- man-made materials
- aesthetics (e.g. visibility)

The order of presentation of the receptors in itself provides an intuitive risk assessment, with health being of highest importance to man.

The value judgments used in delineating between acceptable and non-acceptable effects for standard setting have been provided in Chapter 3. In this portion of the report, the known or suspected spectrum of effects are identified and quantified whenever possible. The cross-comparison of the information in the two chapters provides an indication of the significance of the effect according to various regulatory and scientific agencies.

No value judgments are provided in this section. The effects are documented, but subsequent interpretation will be required to judge their significance. For completeness, any beneficial effects of automotive pollutants are described but no effort has been directed to their quantification.

4.2 Health Effects

4.2.1 Introduction

There is general agreement on the need to protect the public from adverse health effects of automotive air pollutants. However, there is considerable debate on such questions as what constitutes an "adverse" effect, what proportion of the population must be protected from such effects and under what circumstances, and at what exposure levels these effects occur.

It is now well-known that exposure to pollutants generally results in a continuum of responses in the human body. Initially, there may be barely detectable biochemical or morphological alterations of unknown consequence. These functional or structural changes may be viewed as indicative of impending damage or they may be merely adaptive phenomena. In fact what may be regarded as adaptive effects in some individuals may indeed herald impending harm in others, depending often on the presence or absence of concomitant disease or physiological condition. For example, exposure to low levels of carbon monoxide may cause the heart of a healthy individual to work harder, promoting merely a greater "training" effect. Exposure to this same level in an individual with pre-existing heart disease, however, may tip an already compromised cardiac reserve to the point that symptoms of insufficient oxygen (e.g. angina pectoris) may occur. Similarly, exposure to nitrogen dioxide may result in minor increases in airway resistance which may be of no consequence and not even perceptible in some individuals, but the same degree of increased airway resistance may produce breathlessness and inability to perform tasks in other individuals, for example those with chronic lung disease. Moreover, some people are much more sensitive to the effects of some pollutants compared with the general population. There is considerable individual variability, for example to the irritating effects of ozone on the respiratory track. Thus the first point is that small alterations may have different physiological significance in different individuals and the second point is that some individuals are more susceptible to these physiological changes than are others.

Yet a third consideration is that the overall significance of a small impairment varies according to the activity of the exposed individual. While a small decrement in vigilance attributable to carbon monoxide exposures may be of minimal consequence to a person resting on a park bench, it may have enormous impact if the individual is piloting an airplane. Similarly a slightly decreased maximum work capacity would surely go unnoticed in a population of sedentary or mildly exercising individuals, but may be of significance on an urban construction site where total productivity is hampered.

Fourthly, even when an effect is clearly acknowledged as adverse, there are uncertainties as to the exposure levels at which it occurs. For example, there is no disagreement that an exposure (for example, to particulates) which results in increased mortality in the population, is unacceptable. There is also general agreement that increased morbidity, e.g. greater incidence or aggravation of asthma, cardiovascular disease, chronic bronchitis, etc. is of concern. However, as will be illustrated in the following subsections, there is often no widely accepted clear-cut level at which these effects can be said to occur. An understanding of the nature of the available methodology is important to appreciate why this is so.

Evidence regarding the adverse health effects caused by a particular pollutant is derived from three sources. First, there are animal experiments. These are useful to delineate the full range of toxicological effects, to gain insight into potential mechanisms of toxicity and define structure-function relationships between physico-chemical properties of these agents and particular health-effects. It is typically acknowledged that common biochemical events are probably involved for both human and other mammalian species, and the more animal species that respond in a similar manner, the stronger is the basis for qualitative extrapolation of the effect from animal to human beings. However, while some preliminary work is underway to begin to formulate quantitative extrapolations, at present animal experiments can offer only rough guides at best regarding dose-response relationships for humans.

The second body of evidence is derived from controlled experiments on human beings usually in laboratory settings. The lowest concentrations which are shown in these sorts of experiments to cause measurable health effects depend heavily on the particular subjects who are studied. Often these results are not widely generalized to different groups under different conditions. Most experimental human studies are performed on small numbers of relatively healthy persons who may not fully reflect the range of human sensitivity. Moveover, the air to which the subjects are exposed does not include the full mix of chemicals which are in the ambient air, other than the pollutant being studied. Some of these exposures may have an additive effect with the given pollutant in causing adverse health effects. Further, health effects of chronic exposures typically cannot be assessed in human clinical studies.

The third body of evidence comes from epidemiological or community health studies. Since the level of exposure is not under the control of the investigator, these studies can demonstrate important cause-effect relationships, but rarely provide a strong basis for dose-response relationships. Interpretation of exposure data in epidemiological studies has been a major focus of controversy in standard setting. In addition, other factors such as temperature or frequency of smoking have confounded the assessment of air pollution effect, and distinguishing the effect of one pollutant from effects attributable to other pollutants or the combination thereof is often difficult.

Despite these limitations, a large body of information has accumulated regarding the health effects of the various components of automotive emissions. Assessing the data from one source in conjunction with knowledge obtained from other types of studies has enabled the setting of air quality criteria. A review of the state of knowledge follows.

4.2.2 Carbon Monoxide

Introduction and Mechanism of Toxicity

Carbon monoxide (CO) is an odorless, colorless, tasteless, non-irritating gas which is rapidly absorbed through the lungs into the bloodstream. There CO binds with haemoglobin (Hb) to form carboxyhaemoglobin (COHb). As the affinity of Hb for CO is approximately 200-250 times its affinity for oxygen, exposure to even very low concentrations of CO can result in a significant reduction in the oxygen-carrying capacity of the blood. Furthermore, the presence of COHb in the blood shifts the oxyhaemoglobin dissociation curve in such a way that tissue tensions must fall to much lower levels in order for the haemoglobin to release its oxygen. Therefore, exposure to CO not only decreases the oxygen-carrying capacity of the blood, but also impairs the release of oxygen to the tissues, giving rise to a greater degree of oxygen deficiency than would arise from either reduced ambient oxygen tension or an equivalent reduction in Hb resulting from anaemia. As the major effect of exposure to CO is related to this reduced ability of the blood to deliver oxygen, all organs and biological systems for which a continuous high oxygen supply is necessary to maintain function become critical. The three most important of these are the heart, the central nervous system and the fetus (Rylander and Vaslerlund, 1981).

There is also evidence that carbon monoxide blocks the energy flow at the cellular level through the cytochrome system (Goldbaum _et al_., 1975, 1976; Goldbaum, 1977). In any case, it is generally felt (EPA, June 1979; WHO, 1979a) that the hypoxemia mechanism provides an adequate explanation for the observed effects, at least for the purposes of standard-setting.

The principal factors determining the amount of CO present in the body after inhalation of a given concentration of CO for a given duration of exposure include the amount of exercise, body size, diffusion capacity and other health parameters of the lung, barometric pressure and endogenous CO production (i.e. formed from the metabolic breakdown of Hb and other haeme-containing materials). Endogenous production of CO may be significantly increased in persons with haemolytic anaemia, in women during pregnancy and in persons taking certain types of drugs (particularly anticonvulsants), or exposed to certain substances (such as methylene chloride). Population groups most susceptible to CO toxicity are those with high total COHb from endogenous and exogenous sources, as well as those most sensitive to oxygen deprivation. It is generally accepted that individuals with cardiovascular disease, pulmonary disease or anaemia as well as fetuses and pregnant women constitute sensitive populations. Concern exists that because of their increased oxygen requirement from higher metabolic rates, healthy children also consitute a particularly sensitive group (EPA, June 1979; WHO, 1979a). Smokers as well as people occupationally exposed to CO are, of course, also at increased risk. In fact, smoking dwarfs the effect of ambient exposure to CO; moreover it is said that smokers excrete more CO into the air than they inhale from the environment (EPA, June 1979). Therefore, there is no basis for establishing air quality criteria to protect smokers from a possible incremental effect (ibid).

The mathematical model for calculating the formation of COHb at different ambient CO levels and exposure times will be described in a subsequent section.

As discussed in Chapter 3 above, various criteria have been used in the different jurisdiction to arrive at air quality standards. For example, in Canada, the 1971 recommended limits were based on the levels associated with physiological stress on patients with heart disease, impairment of performance in some psychomotor tests, impairment of visual acuity and increased metabolic cost of heavy work. The 1979 U.S. NAAQS for CO was based on the central nervous system effects identified in the study by Beard and Wertheim (1967) (EPA, 1971).

However, on later review, the EPA (Oct, 1979) questioned these findings and based their recent proposed standards on the cardiovascular effect category (EPA, Aug., 1980). The scientific evidence for the various adverse health effects of carbon monoxide will be briefly reviewed in the following sections along with the quantitative assessments that have been attempted in order to provide dose-response relationships.

Deleterious Effects on the Cardiovascular System

Experimental animal studies in numerous species have illustrated the pathological effect of CO on the cardiovascular system (Rylander and Vaslerlund, 1981; EPA, Oct., 1979; WHO, 1979a). Although the significance of some of the animal findings has been the subject of debate, (e.g. Weir and Fabiano, 1982) cardiovascular damage and EKG aberrations have also been demonstrated in studies of both healthy and impaired human subjects (see Table 4.2-1).

The myocardium, or heart muscle, is more sensitive than any other muscle to oxygen lack for a number of reasons. The myocardium depends almost entirely on aerobic metabolism for energy production and has a very low oxygen extraction reserve (James _et_ _al_., 1979). Coronary circulation responds to low oxygen tension by increasing flow rate rather than increasing oxygen extraction. In a person with coronary vascular disease this ability to increase flow rate may be severely curtailed by the presence of atheromatous plaques and therefore the myocardium is forced to try to extract more oxygen, creating a further reduction in coronary oxygen tension. Thus the patient with coronary heart disease who is unable to respond adequately to hypoxia, is particularly vulnerable to the effects of CO, which not only decreases oxygen availability to the heart tissue, but also forces the heart to work harder to try to compensate for oxygen lack to other tissues. It therefore seems appropriate that persons with cardiovascular disease (CVD) have been designated as the group at greatest risk from low-level ambient exposure to CO (e.g. EPA, Aug., 1980).

TABLE 4.2-1

EFFECTS OF CO EXPOSURE ON THE CARDIOVASCULAR SYSTEM IN HUMANS

Selected References	COHb level% (final means)	Exposure	Effects
A. Aggravation of Angina			
Aronow, 1978	1.77	2-h, tobacco smoke	Duration of exercise until onset of angina decreased 22%
Aronow, 1981	2.02	1-h, 50 ppm CO ($57.5\ mg/m^3$)	Duration of exercise until onset of angina decreased 10%
Aronow, 1978	2.28	2-h, tobacco smoke	Duration of exercise until onset of angina decreased 38%
Aronow and Isbell, 1973	2.68	2-h, 50 ppm CO ($57.5\ mg/m^3$)	Duration of exercise until onset of angina decreased 16%
Anderson et al., 1973	2.9 and 4.5	4-h, 50 ppm and 100 ppm CO (57.5 and $114.5\ mg/m^3$)	Duration of exercise until onset of angina decreased 15% Duration of pain increased 31%
Aronow et al., 1972	5.08	1-h, 42-63 ppm CO, freeway (48.1-$72.1\ mg/m^3$)	Duration of exercise until onset of angina decreased 33% 15% after 2 hours; systolic blood pressure and heart rate at angina also significantly decreased
B. Morbidity and Mortality from Myocardial Infarction			
Kurt et al., 1979	0.6-0.9	estimated	Association between ambient CO levels and frequency of initial cardiorespiratory complaints at an emergency room at a Denver hospital

TABLE 4.2-1 (CONT'D)

Selected References	COHb level% (final means)	Exposure	Effects
Cohen et al., 1969	estimated 1.4	Weekly mean of 9 ppm (10.3 mg/m^3)	Significant difference in fatality rates between high and low pollution areas
Kuller et al., 1975	estimated 1.6	7.7-14 ppm (8.8-19.5 mg/m^3)	No relationship established in the Baltimore area between ambient CO levels and onset of sudden death or myocardial infarction
Hexler and Goldsmith, 1971	3.5	7.3-20.2 ppm 24 h. mean (8.4-23.1 mg/m^3)	A greater number of deaths occurred in L.A. when ambient CO concentrations were higher
C. Aggravation of Peripheral Vascular Disease			
Aronow et al., 1974	2.77	2-h, 50 ppm (57.5 mg/m^3)	Aggravation of intermittent claudication
D. Effects on Work Performance and other Cardiovascular Effects			
Drinkwater et al., 1974	3.95		Reduced work time for exposed nonsmokers, no reduction in exposed smokers
Aronow et al., 1977	4.08	1-h, 100 ppm (114.5 mg/m^3)	Reduced exercise time until breathlessness in patients with chronic obstructive pulmonary disease
Aronow and Cassidy, 1975	estimated 4.08	1-h, 100 ppm (114.5 mg/m^3)	Reduced exercise time until exhaustion in middle-aged healthy nonsmokers

TABLE 4.2-1 (CONT'D)

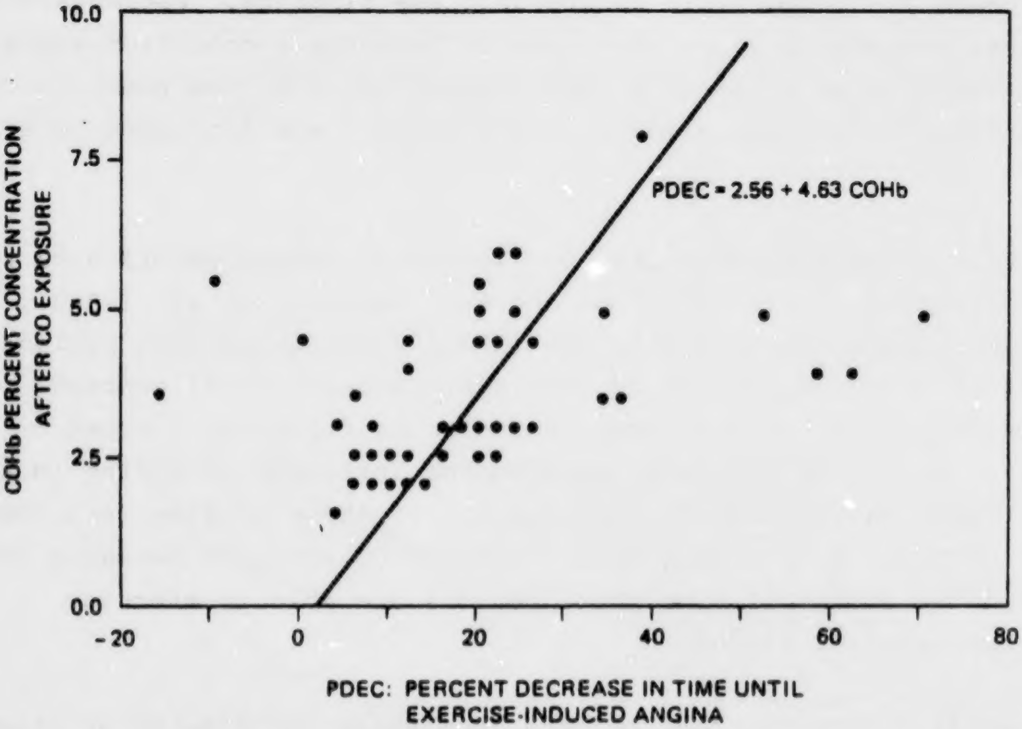
Selected References	COHb level% (final means)	Exposure	Effects
Ayres *et al.*, 1970	5-25	8-15 min. 1000 ppm (1145 mg/m^3)	Increased cardiac output and coronary blood flow. Signs suggesting myocardial hypoxia in patients with CVD
Ayres *et al.*, 1969	8.96	8-15 min. 1000 ppm (1145 mg/m^3); 30-45 sec, 5000 ppm (5725 mg/m^3)	Increased coronary blood flow and decreased extraction ratios. Increased system's oxygen extraction
Ekblom and Huot, 1972	5-20	15 min.	Decreased maximal exercise time and maximal oxygen uptake with increased COHb levels in healthy adults

The effect of low levels of CO on angina pectoris (a symptom of CVD in which mild exercise or excitement produces a sense of pressure or pain in the chest due to insufficient oxygen supply to the myocardium) has received considerable attention by scientists and regulators, who have concluded that aggravation of angina pectoris definitely constitutes an adverse health effect (e.g. EPA, June 1979) attributable to CO. Some epidemiological studies have suggested that increased mortality from myocardial infarction (death of heart tissue, commonly known as "heart attack") is associated with increased CO exposure, although there is considerable debate on these findings. Patients with peripheral vascular disease also seem to have their condition aggravated by CO. The studies relating to the effects of CO on the human cardiovascular system are summarized in Table 4.2-1 and discussed briefly below.

Aggravation of Angina Pectoris: Two groups of investigators have reported effects of low level CO on angina. Anderson et al. (1973), Aronow and Isbell (1973) and Aronow (1981) directly exposed patients with angina to CO in laboratory settings. Aronow (1978) exposed subjects in ventilated and unventilated rooms filled with tobacco smoke, and Aronow et al. (1972) studied the influence of riding in an open car on a major freeway. The results of these studies (see top section of Table 4.2-1) show that low level CO exposure causes a decrease in the amount of time these persons are able to exercise before the onset of angina.

Keeney et al. (1982) plotted the data from these experiments as shown in Figure 4.2-1. Using linear regression the authors calculated that for each percent increase in COHb level, the time until onset of angina increases on average of 4.63%. As there was considerable variation (correlation coefficient, R^2, only 0.166) this relationship was not used in their subsequent risk assessment. What is clear, however, is that COHb levels as low as 2.5-3% can definitely aggravate angina. The possibility of effects at COHb levels of 2% or even lower has been suggested (Aronow, 1981) but awaits confirmation.

FIGURE 4.2 -1 COHb level and time until exercise-induced angina.*



* Reproduced from Keeney et al., 1982
(The authors excluded Aronow's 1978 tobacco smoke study due to the presence of other pollutants. They did, however, include the freeway study conducted by Aronow et al. in 1972, although confounding factors may also have been problematic.)

Since sufficient data to provide numerous points on a dose-response curve were not available from carefully controlled human experiments, the EPA has developed a technique in which experts are interviewed as to their opinion based on information from clinical and epidemiological studies as well as professional experience and judgement (Keeney et al. 1980). The assessment of the experts for a dose-response relationship depicting the reduction in time until onset of angina at given COHb levels is provided in Table 4.2-2 and Figure 4.2-2. The percent of angina patients with an "aggravated condition" is defined as those experiencing at least a 10% reduction in time until the onset of pain. The assessment of the experts regarding the percent of angina patients that would probably suffer additional attacks at given COHb levels is shown in Table 4.2-3. and Figure 4.2-3. The correlation coefficient for the model is 0.99, indicating a high degree of consensus. The data indicated that at a COHb level of 3.5%, 56.1% of angina patients may be expected to suffer additional attacks (with 95% confidence intervals ranging from 32.1% to 77.7%).

Increased Risk of Myocardial Infarction: Several epidemiological studies (Cohen et al., 1969; Goldsmith and Landaw, 1968; and Hexter and Goldsmith, 1971) conducted in the Los Angeles area have suggested the possibility of increased mortality from myocardial infarction in areas with high ambient levels of CO (sufficient to produce COHb levels of 8-17%) compared to those in less CO polluted areas. There has been considerable controversy regarding the interpretation of these results as hospital admission rates did not correlate with CO levels, and some investigators (eg. Cohen et al., 1969) did not measure COHb levels and failed to control for smoking and occupation. A similar study in Baltimore (Kuller et al., 1975) failed to find such a correlation albeit at considerately lower ambient CO levels (sufficient to produce COHb levels in the range of 1-10%).

Table 4.2 -2 DOSE-RESPONSE RELATIONSHIP BETWEEN COHb LEVELS AND PERCENTAGE OF ANGINA PATIENTS SUFFERING ADDITIONAL ATTACKS *

Aggregated Data		Assessed Expert Data P_A **		
COHb (percent)	P_A^*(percent)	Aronow	Kurt	Mustafa
1.0	2	0-5(2 median)		
1.5	10	5-20(10 median)		
2.0	20	10-40(25 median)	20	10-20
2.5	35	40	30	
4.0	60	60		
5.0	80			

* Reproduced from Keeney _et al_., 1982.
Data derived from the opinion of experts as outlined in the report
** Percentage of angina patients suffering additional attacks

Figure 4.2-2 Dose-response data and curve for the percentage of additional angina attacks to angina patients. *

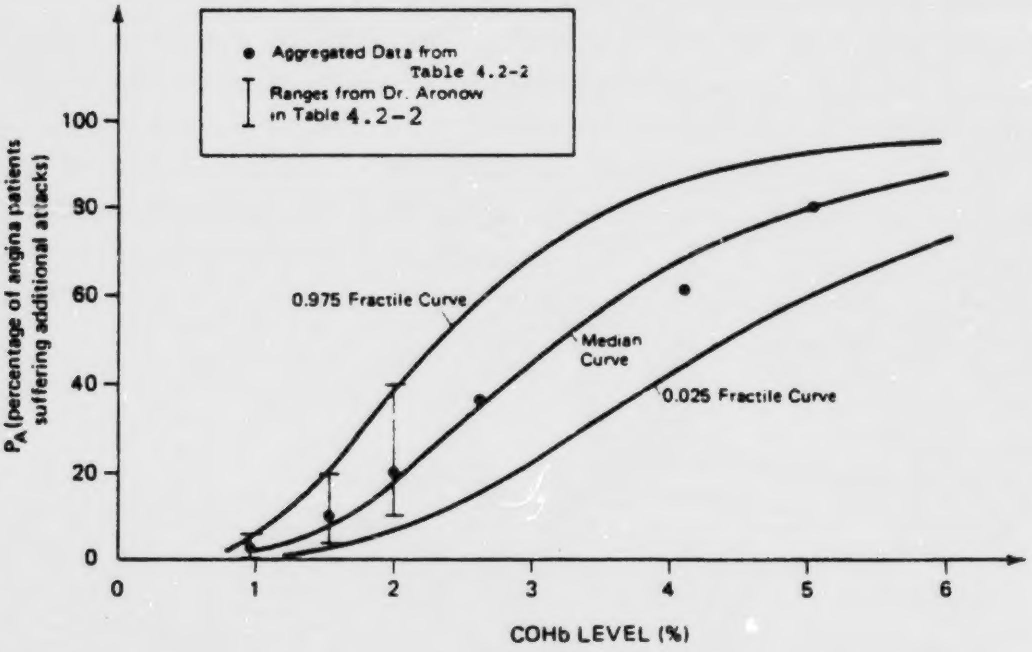
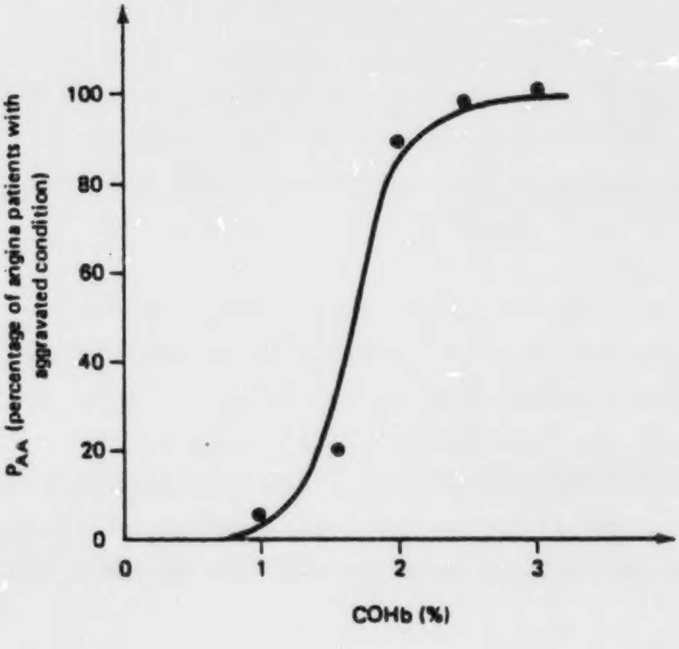


Table 4.2-3 DOSE-RESPONSE RELATIONSHIP BETWEEN COHb LEVELS AND PERCENTAGE OF ANGINA PATIENTS WITH AGGRAVATED CONDITION*

COHb (percent)	P_{AA}** (percent)
1.0	5
1.5	20
2.0	90
2.5	99
3.0	100

* Reproduced from Keeney *et al.*, 1982
Data based on the opinions of experts as outlined in the report.
** Percentage of angina patients with aggravated condition

Figure 4.2-3 Dose-response data and expected dose-response curve on relating COHb levels and the percentage of angina patients with aggravated condition. *



Thus the possibility of an association between ambient CO levels and incidence and/or fatality from myocardial infarctions remains a source of contention. However, if confirmed, it is interesting to note that Keeney *et al.* (1982) have calculated from the Hexter and Goldsmith (1971) data that when the CO level changes from the minimum observed level of 8.0 mg/m^3 (7.0 ppm) daily mean to the maximum of 23.1 mg/m^3 (20.2 ppm) daily mean, there would be an estimated 11 more deaths/day in L.A. County (based on 1962-1965 mortality rates).

The dose-response relationship describing the level of COHb associated with percent increase in total heart attacks (fatal and non-fatal) among those with heart disease, derived from judgement of the experts, is shown in Table 4.2-4 and Figure 4.2-4. It is noted that the fit is excellent and that the standard deviation (i.e. variability of response) is greater at the higher COHb levels. If these estimations are confirmed, they might well set the ultimate limit to urban CO exposure.

Aggravation of Peripheral Vascular Disease: The one clinical study (Aronow *et al.*, 1974) to examine such an effect, involved the exposure of 10 persons with occlusive arterial disease to 55 mg/m^3 (50 ppm) CO for 2 hours, followed by exercise until leg pain ("intermittent claudication"). Exposure sufficient to produce COHb levels of 2.8% significantly decreased the time until onset of pain and cessation of activity.

Other Cardiovascular Effects: Ayres *et al.* (1969, 1970) have shown that blood flow indeed increases as a compensatory response to CO exposures. This finding has relevance with respect to coronary damage or other vascular effects that may result from the cardiovascular system being pushed beyond its capacity.

Finally, reference is often made to the study of Kurt *et al.* (1979) in which the cardiorespiratory complaints of patients at a Denver emergency room were evaluated on "high CO days" (average 31 mg/m^3; 27 ppm) as compared to "low CO days" (average 14 mg/m^3; 12 ppm). The significance of the positive findings are questionable as: (1) the cardiorespiratory complaints in the study could not serve as specific indicators of cardiovascular damage, (2) the authors did not report

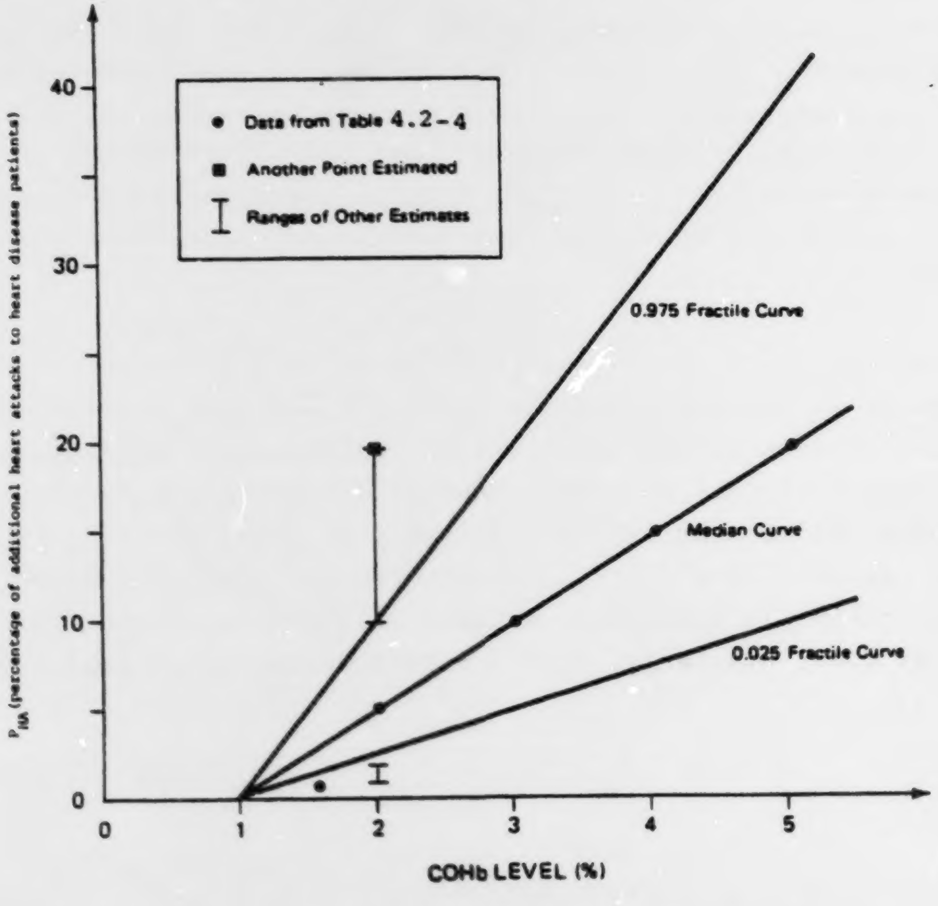
Table 4.2-4 DOSE-RESPONSE RELATIONSHIP BETWEEN COHb LEVELS AND PERCENTAGE INCREASE IN HEART ATTACKS*

COHb(percent)	P_{HA}(percent)**
1.5	1
2.0	5
3.0	10
4.0	15
5.0	20

* Reproduced from Keeney et al., 1982.
Data based on the opinion of experts as outlined in the report.

**P_{HA} Percentage of additional heart attack to heart disease patients.

Figure 4.2 -4 Dose-response data and curve for the percentage of additional heart attacks to heart disease patients*



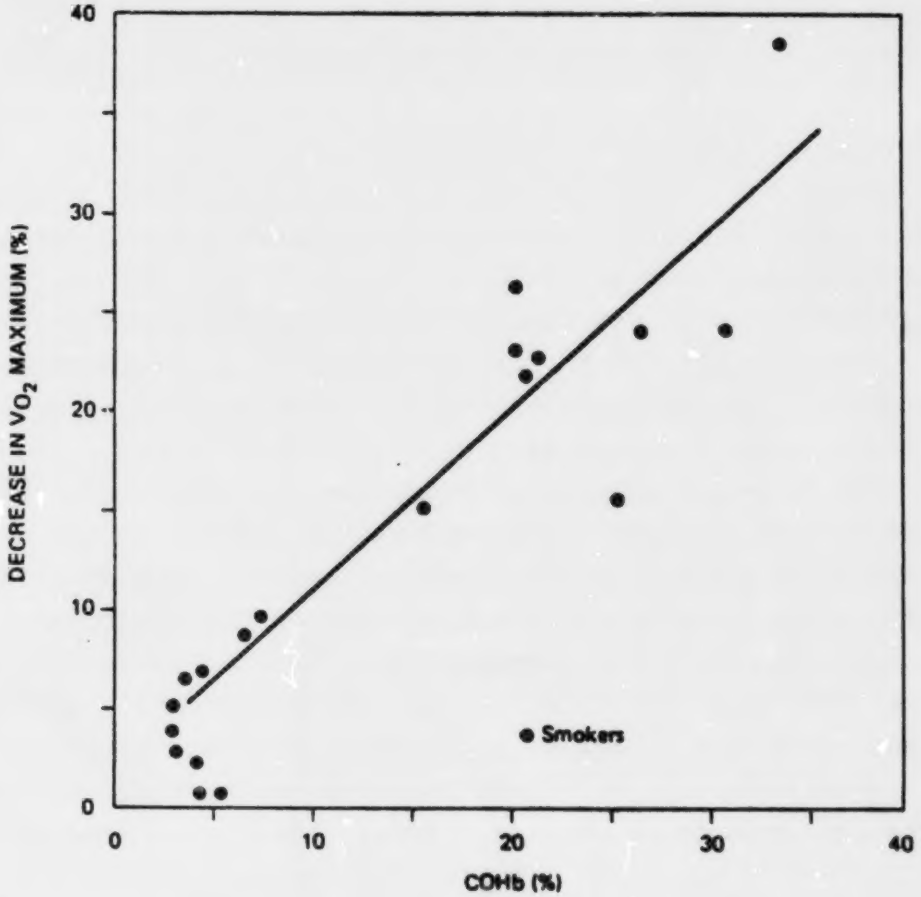
any COHb levels and (3) the single monitoring site near the hospital could not be considered an adequate indicator of the exposure sustained by the patients.

Effects on Work Capacity and Pulmonary Function

Several studies (including those conducted by Ekblom and Huot, 1972; Horvath et al., 1975 and Ayres et al., 1970) have indicated that COHb levels in the range of 5-33% can produce a linear decline in maximum oxygen uptake and hence work capacity of healthy individuals (see Figure 4.2-5). Other studies suggest that work capacity, may not be affected by COHb levels of 10-20% for short periods of submaximal exercise (see EPA, June, 1979). Aronow and Cassidy (1975) actually reported decreased exercise performance in normal persons at COHb of 3-6.5% and Aronow et al. (1977) revealed decreased exercise performance in patients with chronic obstructive pulmonary disease (COPD) at this level. While persons with COPD (e.i. asthma, emphysema and chronic bronchitis) are presumably at high risk of CO toxicity due to their pre-existing low oxygen levels, it has been suggested that these persons may absorb less CO due to their disease and may have developed other compensatory adaptations such as increased red blood cell production. Aronow et al. (1977) concluded that the 33% reduction in time until exercise-induced dyspnea was seen in these patients after CO exposure was probably a cardiovascular limitation rather than a respiratory one.

From the EPA's interviews with the scientific authorities on these issues it was noted (Keeney et al., 1982) that most experts believe that CO does indeed aggravate the condition of patients with respiratory disease. Aronow estimated that 5% of patients with COPD will experience health effects at 1% COHb, 20% at 1.5% COHb, 90% at 2.0% COHb and 99% of patients have aggravation of COPD at 3.0% COHb. Ayres estimated that seriously ill pulmonary patients will experience health effects at 2.0 - 4.0% COHb. Similarly, Kurt and Collier estimated that 2.0% COHb will cause

Figure 4.2-5 **Relationship between COHb and decrement in maximum aerobic power. ***



*Reproduced from E.P.A., Oct. 1979.
The data was obtained from the studies by Horvath and others. It is noted that the linear relationship does not apply to smokers, who in Horvath's series, had COHb levels considerably in excess of 4-5% with no decrement in their respective maximum aerobic power.

dyspnea in these patients, limiting their activity. Mustafa estimated that 10-20% of emphysema patients will have a 20-50% reduction in activity at 2.0% final COHb, as well as 5-10% of bronchitic patients. He felt that the chance of dying during a serious asthma attack will increase by 5-10% at 2.0% COHb. Rokan agreed that severe emphysema patients are most vulnerable to CO but did not feel that asthma attacks would be aggravated by low levels of CO. Crandall agreed that CO could aggravate COPD patients but did not feel that these effects would be widespread below 2.5% COHb for short durations. Benignus did not feel that CO had an important effect on COPD patients.

Deleterious Effects on the Nervous System (CNS)

There has been much controversy regarding the effect of low levels of COHb on subtle CNS functions. This is largely due to the many complex methodological considerations such as providing control periods so that task - learning effects do not mask results, providing double-blind administration, and controlling for confounders (sensory deprivation, drugs, temperature, time of day, etc.) As the CNS has a high oxygen consumption it would logically be expected to be one of the critical organs for CO toxicity. The various CNS functions that have been investigated can be divided into two broad categories. One consists of vigilance tests (ability to detect small changes in the environment that take place at unpredictable times). These include estimation of short-term intervals, reaction to visual or auditory stimuli, discrimination between different visual or auditory signals and maintaining attention or concentration. The second is motor tests, which consist of tests of the performance of physical tasks. Results of some of these studies are listed in Table 4.2-5. While some investigators found effects at 2% COHb (Beard and Wertheim, 1967) others failed to detect impairment at levels above 5% to even 20% COHb (eg. Stewart et al., 1973; and Winneke, 1974). On carefully evaluating the literature

TABLE 4.2-5

SUMMARY OF REPORTED CHANGES IN VIGILANCE AND OTHER CENTRAL NERVOUS FUNCTIONING IN SELECTED STUDIES OF HUMAN EXPOSURE TO CARBON MONOXIDE*

Reference	COHb Level	Effect	Comment
Beard and Grandstaff (1972)	1.8	Impaired vigilance task; increased errors in time estimation.	Possibly exaggerated by sensory isolation. Effects reversed at higher dose. Possible peer pressure effect.
Beard and Grandstaff (1975)	1.8 - 7.5	Impaired visual vigilance.	Bigger decrements in vigilance at 1.8% and 5.2% COHb from at 7.59%.
Fodor and Winneke (1972)	2.3 - 3.1 estimated	Impaired vigilance task (auditory).	Effect disappeared with continuation of experiment.
Beard and Wertheim (1967)	2.5 - 4.0 estimated	Impaired auditory duration discrimination.	Possible sensory isolation effect.
Beard and Wertheim (1969)	2.5 - 4.0 estimated	Impaired time perception.	as above
Groll-Knapp et al (1972)	3.0 - 7.6 estimated	Impaired vigilance task (weaker auditory tones).	Dose-related decrements in vigilance.
Beard and Grandstaff (1970	3.0	Impaired visual intensity discrimination.	Possible sensory isolation effect.
Milulka et al. (1971)	3.0	Impaired tracing task.	Possibly statistically unreliable.
Ramsey, 1972	3.5 - 4.0	Impaired choice reaction time.	Many negative results for simple reaction time.
Horvath et al (1971)	5.0	Decreased vigilance (visual).	Decrements in vigilance for the group with final COHb of 6.6% no effect at COHb of 2.3%.
Winneke (1974)	5.1 - 10%	No effect on auditory vigilance.	
McFarland (1973)	6 -17%	Decrements in peripherial vision reported but no effect on driving performance.	
Bender et al (1971)	7.2	Impaired mental performance and finger dexterity.	
Stewart et al (1970)	15 - 20%	No decrements in time estimation, but delayed headaches and decreased coordination.	
Stewart et al (1973)	15 - 20%	No decrements in time estimation even with levels up to 20% COHb.	
Ray and Rockwell (1970)	10, 20%	Increase in reaction time to taillight intensities and relative speeds but decrease in accuracy of maintaining a fixed distance between cars.	

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* This table was adapted from the data contained in EPA, Oct., 1979; W.H.O, 1979a; Keeney et al, 1982; and Shephard, 1983. For a more complete list of studies of this nature see these sources.

the EPA concluded that CO exposures capable of producing 4-6% COHb can produce adverse effects on the CNS. This conclusion was based largely on the work of Horvath et al. (1971), Groll-Knapp et al. (1972) and Fodor et al. (1973) with respect to impaired vigilance. The findings of Bender et al. (1971) which showed a statistically significant diminution of visual perception, manual dexterity or ability to learn at COHb concentrations of 5-20% were also cited. The EPA concluded that visual sensitivity might be affected as a continuous dose-response function with no obvious threshold, but that confirmatory data was required. In the Swedish review of the literature with respect to standard setting (Rylander and Vaslerlund, 1981) it was concluded that no deleterious effects on the CNS have been demonstrated at COHb below 5%.

The EPA (Aug., 1980) has pointed out that vigilance and visual function effects are of considerable importance since these functions are components of more complex tasks, such as driving, and reduced vigilance could lead to increased accidents. Numerous studies (as summarized by EPA, Oct. 1979; WHO, 1979a; Rylander and Vaslerlund, 1981; and Shephard, 1983) indeed suggest that the performance of complex tasks is adversely affected by low-level CO. Shephard (1983) specifically points out that the "on-the-road" studies of Weir et al. (1973), McFarland (1973), Ray and Rockwell (1970), Rockwell and Ray (1967) and Rockwell and Weir (1975) support the view that CO exposure can affect road safety. There indeed is some suggestive (but not conclusive) evidence implicating CO in fatal motor vehicle accidents (eg. Yabroff et al., 1974) , as noted by the E.P.A. (Aug., 1980). However, other investigators failed to note such a correlation (Ury et al., 1972). Shephard (1983) also noted that in some aircraft accidents, CO poisoning has been implicated, as a result of faulty oxygen equipment, cabin contamination by exhaust fumes, smoking and fires (Blackmore, 1974; Howlett and Shephard, 1973).

Deleterious Effects on Fetal and Neonatal Development

The rate of endogenous production of CO is known to be increased in pregnancy. Longo (1977) attributes about 15% of this increase to CO production by the fetus and another 30-40% to the increased number of red blood cells in the mother. Fetal uptake of CO takes place more slowly than maternal uptake, but after a few hours the fetal COHb level significantly exceeds that of the mother. Moveover, a given COHb level has greater detrimental effects on the fetus. The fetal oxygen tension is probably close to critical values and the fetus is unable to mount the same compensatory mechanisms (increased cardiac output, coronary blood flow and tissue blood flow) as can the adult. The potential of CO to interfere with fetal tissue oxygenation during important developmental stages is therefore of concern. Table 4.2-6 summarizes some of the experimental animal studies which link low level CO exposure to such outcomes as increased perinatal mortality rate, decreased birth weight, decreased weight gain, and lower behavioral activity levels.

The hypothesis that CO exposure during pregnancy affects learning and social behaviour development in humans is supported by studies examining the impact of maternal smoking and altitude on the unborn child. As summarized in Table 4.2-7, decreased birth weight, increased perinatal deaths and increased incidence of congenital heart disease have been linked with increased COHb levels in smoking mothers. Reduced reading attainment in later childhood, as well as decreased general ability, reading and mathematics have also been reported (see Shephard, 1983 and Rylander and Vaslerlund, 1981). The fact that cigarettes contain substances other than CO precludes any direct quantitative application of the results. Although acknowledging the extreme sensitivity of the rapidly growing fetal tissues to CO toxicity, the Swedish authorities, in their careful evaluation of this issue, felt that COHb levels in excess of WHO standards would likely be required for the occurrences of any significant effects (Rylander and Vaslerlund, 1981). The EPA (Aug, 1980) felt that the evidence regarding fetal effects should come to bear on the selection of a margin of safety.

Quantitative Assessments

Sensitive Populations: Some of the conditions giving rise to increased endogenous production of CO were alluded to above and are summarized in

Table 4.2-6 Fetal effects of maternal carbon monoxide exposure: animal experiments. *

	CO/COHb level (exposure duration)	Animal model	Effects	Investigator Year
**	90 ppm, 9--10 % COHb (30 d continuous)	pregnant rabbits	litters of exposed mothers had significantly lower birthweights; great increase in stillborn births and number of neonates who died within first 24 h; no differences in mortality between exposed and control groups at days 6 and 21; some neonates born without a leg.	Astrup 1972
	0.1—0.3 % inspired CO (1--3 h)	pregnant rhesus monkeys	pregnant mothers sustained up to 60 % COHb without clinical sequalae; fetuses whose arterial oxygen content fell below 2.0 ml/100 ml for at least 45 min showed severe brain damage.	Ginsberg and Myers 1974
	0.1—0.3 % inspired CO (exposed until fetuses obtained a »moderate« or »severe« hypoxia + 1 hour)	pregnant rhesus monkeys	widespread cerebral necrosis in fetuses whose arterial oxygen content fell to 1.6--1.8 ml/100 ml.	Ginsberg and Myers 1978
	15 % COHb (entire pregnancy)	pregnant rats	no differences in number of offspring per litter, mortality rate at day 1 or any gross teratologic effects; insignificant difference in birthweight between exposed and controls, which became significant at day 4; markedly lower brain protein levels in CO neonates; changes in CHS in connection with prenatal CO exposure.	Fechter and Annau 1977
	30 or 90 ppm or low oxygen (continuous exp)	pregnant rats	great reduction in number of successful pregnancies; fetuses exposed to low O_2 showed increased hematocrit; no differences in number of live, recently dead or visibly abnormal fetuses.	Garvey and Longo 1978
** **	30, 50, 100 ppm (24—48 h), 300 ppm (2—3 h)	pregnant sheep	fetal COHb levels rose more slowly than maternal, took longer to wash out, and were at maximum considerably higher than maternal levels.	Longo and Hill 1977
**	250 ppm, 10—15 % COHb (7 or 24 h/d, 6—15 or 6 -18 d of gestation)	pregnant rabbits and mice	mean body weight of mice fetuses higher in 7 h/d group and lower in 24 h/d group than controls; 2 of 18 in 24 h/d group had malformations; exposed had more lumbar ribs and spurs than controls.	Schwetz *et al.*, 1979

** Mg/M^3 Conversions:

ppm	30	50	90	100	250	300
mg/m^3	34	57	103	115	286	344

Table 4.2 -7 Fetal effects of maternal carbon monoxide exposure: studies in humans *

Investigators Year	Description	Effects
Astrup *et al.*, 1972	smoking habits and COHb levels of 253 pregnant women recorded; birthweights and conditions of neonates later recorded.	mean birthweight of neonates of non-smoking mothers was 3,225 g. and of smoking mothers 2,990 g
Goujard *et al.*, 1975	prospective investigation of 6,989 pregnant women.	250 % increase observed in number of stillbirths among smoking mothers; a large proportion of the increase was due to abruptio placentae.
Meyer *et al.*, 1976	further analysis of data collected in Ontario Perinatal Mortality Study.	neonatal birthweight and length of gestation seen to be directly related to mothers' smoking habits; placental complications increased with the level of smoking, except for mothers pregnant for the first time smoking less than 1 pack/day, particularly for placenta previa and abruptio placentae.
Fedrich *et al.*, 1971	further analysis of data collected in 1958 British Perinatal Mortality Study and National Child Development Study, with reference to identifying possible connections between maternal smoking and incidence of congenital heart disease.	analysis demonstrated that the occurrence of congenital heart disease in babies was 50 % greater among smoking mothers than non-smoking mothers; sample too small, however, to allow for firm conclusions.
Buncher 1969	data collected from obstetrical wards in Naval hospitals in U.S.; effects of smoking during pregnancy studied.	observed difference in length of pregnancy between non-smoking and smoking mothers (more than 1 pack/day) was 29 to 24 h; it was suggested that about 10 % of the known reduction in birthweight could be attributed to this shortened gestation period.

*Reproduced from Rylander and Vaslerlund, 1981. See source for complete references.

TABLE 4.2-8

CONDITIONS ASSOCIATED WITH INCREASED SENSITIVITY TO CO EXPOSURE

Increased endogenous production - pregnancy, newborn, haemolytic disease, saturation dives, drugs (e.g. phenobarbital, diphenyl hydantoin), toxic substances (e.g. methylene chloride)

Hypoxic conditions - fetus, anaemia, chronic obstructive lung disease, cardiovascular disease, peripheral vascular disease, cerebral vascular disease, high altitude residents, elderly

Hypermetabolic conditions - increased ventilation due to vigourous physical work, pregnancy, high altitude, thyrotoxicosis, newborn infants, hypoxic conditions

Table 4.2-8, along with other hypoxic conditions and hypermetabolic conditions that increase sensitivity to CO toxicity. The interaction between high altitude and CO exposure may be an important public health consideration in the Canadian Rockies. At moderate altitudes, an ascent of 300 meters may be equated to a 1% increase of blood COHb content. Thus people living above 100 meters are particularly vulnerable to low levels of CO.

For tobacco smokers, the primary source of CO and the resulting COHb levels is from intake of tobacco smoke (Coburn et al., 1965). As mentioned earlier, it has been noted (EPA, June 1979) that smokers excrete CO into the air rather than inhale it from the environment and are unable to absorb further CO at normal ambient pressures. As Shephard (1983) notes, adaptation seems to occur with chronic exposure to high concentrations of CO, in any case. Similarly, Shephard (1983) notes that workers occupationally exposed to CO are generally also unable to absorb further CO from the atmosphere and ambient air. In the case of these workers, concentration of CO is of significance only in so far as it restricts elimination of occupationally absorbed CO. Thus, smokers and workers occupationally exposed to CO are generally not considered in setting air quality standards for the population as a whole.

CO Exposure and Resulting COHb levels: In 1912 Haldane and his co-workers (Douglas et al., 1912) developed an equation relating CO exposures and equilibrium COHb levels. The time required to reach equilibrium, however, is influenced by a number of factors, the most important for healthy individuals being the level of exercise. At rest, approximately 8-12 hours are needed to achieve equilibrium; for a moderate walk (∿ 3 miles/hr) equilibrium can be reached in half the time. Coburn et al. (1965) therefore developed an equation to permit calculation of COHb at given ambient levels of CO as a function of time, considering appropriate physiological parameters. Experimental data obtained by Peterson and Steward (1970) largely confirm the accuracy of this model. Table 4.2-9 illustrates the application of the Coburn model. A moderate level of exercise, equivalent to a 3 mile/hr walk was selected by the EPA as a reasonable estimate of the maximum exercise

TABLE 4.2-9

PREDICTED COHb RESPONSE TO 1-h AND 8-h EXPOSURE TO CONSTANT CO CONCENTRATIONS AT LIGHT AND MODERATE ACTIVITY, BASED ON COBURN EQUATION AND STANDARD PARAMETERS*

CO		Exposure time (hours)			
		1		8	
mg/m³	ppm	Intermittent Rest/ Light Activity	Moderate Exercise	Intermittent Rest/ Light Activity	Moderate Exercise
8.0	7.0	0.7	0.7	1.2	1.2
10.3	9.0	0.7	0.8	1.4	1.5
13.7	12.0	0.8	1.0	1.8	1.9
17.2	15.0	1.0	1.1	2.2	2.4
22.9	20.1	1.1	1.4	2.9	3.1
28.6	25.0	1.3	1.6	3.6	3.8
40.1	35.0	1.6	2.1	4.9	5.3
57.3	50.0	2.2	2.9	7.0	7.6

* Assumed parameters: Alveolar ventilation rates: intermittent rest/light activity= ventilation rate of 10 litres/min, moderate activity=ventilation rate of 20 litres/min (equivalent to 3 mph walk on level ground or light industry or housework); Haemoglobin=15 g/100 ml (normal male); altitude=sea level; initial COHb level=0.5 percent (normal level due to endogenous CO production).

level achieved by most persons with cardiovascular disease (EPA, June, 1979). At this level of activity, 1-h exposure to 40 mg/m^3 (35 ppm) would lead to 2.1% COHb, and 8-hours exposure to 10 mg/m^3 (9 ppm) would result in 1.5% COHb. At rest, the 1-and 8-hr standards are reasonably consistent in that both result in about 1.4-1.6% COHb. As the importance of exercise has been increasingly recognized recently, it has been recommended to drop the 1-hour standard to 29 mg/m^3 (25 ppm) so as to make the exercising 1-and 8-hr standards consistent.

However, uncertainties exist in using constant CO concentrations to estimate expected COHb levels as presented in Table 4.2-9. Biller and Richmond (1982) point out that variations in physiological parameters used in the Coburn model are sufficient to provide noticeable deviations from the COHb levels in Table 4.2-9 and that predictions based on exposure to constant CO concentrations inadequately represent widely fluctuating concentrations in ambient air. Ott and Mage (1978) indicated that the assumption of a constant concentration for an 8-hour averaging time can lead to underestimating the COHb response to CO concentrations that are actually fluctuating on a much shorter time scale. Biller and Richmond performed a sensitivity analysis using the Coburn model in a dynamic mode for individuals with physiological parameters different from the baseline values of Table 4.2-9. The results of their analysis are given in Table 4.2-10. The ranges that appear in the 8-hour portion of this table are the ranges of COHb values obtained from 20 different sets of CO data. Comparison of the baseline numbers for the 8-hour case in Tables 4.2-9 and 4.2-10 illustrates that the upper end ranges of the dynamic calculations is above the values of the static calculation.

Health Effects at Various Ambient Levels of CO: The health effects expected to occur at various levels of CO are summarized in Table 4.2-11. Shephard (1983) has pointed out that the critical health effect of exposure to CO observed in the general population is a deterioration of vigilance. However, individuals most vulnerable to CO, patients with cardiovascular and peripheral vascular disease, experience adverse health effects at lower levels of COHb, possibly as low as 2%. It is, of course, recognized that individuals with higher endogenous productions will achieve a given level of COHb at lower ambient CO levels. The importance of setting margins of safety was discussed in Chapter 3.

TABLE 4.2-10

PREDICTED COHb RESPONSE TO 1-h AND 8-h EXPOSURE TO CO AT VARIOUS ACTIVITY LEVELS, BASED ON COBURN EQUATION BUT USING DYNAMIC CO CALCULATIONS AND VARIABLE PARAMETERS *

COBURN MODEL ESTIMATES FOR CARBOXYHEMOGLOBIN LEVELS ASSOCIATED WITH ALTERNATIVE ONE-HOUR CARBON MONOXIDE STANDARDS LEVELS[a]

Standard Level	COHb Levels (%) Predicted for a Range of Ventilation Rates for Exposure to the Specified Standard Concentration Level for 1-Hour[b]			
	Case 1	Case 2	Case 3	Case 4
	Baseline (nominal) physiological parameters	High range of physiological parameters for normal persons		Typical hemolytic anemic persons
(ppm)		@ Sea level	@ 5000 ft	
20	1.1 - 1.3	1.7 - 2.1	1.7 - 2.1	2.5 - 2.7
25	1.2 - 1.5	2.0 - 2.5	2.0 - 2.5	2.8 - 3.0
35	1.5 - 2.0	2.6 - 3.3	2.6 - 3.3	3.4 - 3.7

[a] A daily maximum standard with one expected exceedance per year.

[b] Coburn model parameters:

All Cases: Ventilation rate is 10 liters/min at lower end and 20 liters/min at upper end of range.

Case 1: As given in Table 4.2-9

Case 2: Hemoglobin = 13 g/100ml; initial COHb = 0.7%; endogenous rate = 0.014 ml/min; blood volume = 3500 ml; CO lung diffusivity = 40 ml/min/torr; Haldane constant = 246.

Case 3: Same as for Case 2, except for difference in altitude.

Case 4: Hemoglobin = 9 g/100ml; initial COHb = 1.9%; endogenous rate = 0.03 ml/min; blood volume = 4600 ml; CO lung diffusivity = 25 ml/min/torr; Haldane constant = 218.

mg/m³ Conversions:								
	ppm	7	9	12	15	20	25	35
	mg/m³	8	10	14	17	23	29	40

COBURN MODEL ESTIMATES FOR CARBOXYHEMOGLOBIN LEVELS ASSOCIATED WITH ATTAINMENT OF ALTERNATIVE EIGHT-HOUR CARBON MONOXIDE STANDARD LEVELS[a]

Standard Level	Maximum COHb Levels (%) Predicted on a Day when 8-Hour CO Concentration Just Attains Standard Level, for a Range of Actual Air Quality Patterns Adjusted to Simulate Attainment of the Specified Standard[b,c]			
	Case 1	Case 2	Case 3	Case 4
	Baseline (nominal) physiological parameters	High range of physiological parameters for normal persons		Typical hemolytic anemic persons
(ppm)		@ Sea level	@ 5000 ft	
7	1.1 - 1.4	1.5 - 1.9	1.7 - 2.1	1.8 - 2.2
9	1.3 - 1.8	1.9 - 2.4	2.1 - 2.6	2.1 - 2.6
12	1.7 - 2.3	2.4 - 3.2	2.6 - 3.4	2.6 - 3.3
15	2.1 - 2.8	2.9 - 3.9	3.1 - 4.1	3.0 - 3.9

[a] A daily maximum standard with one expected exceedance per year.

[b] COHb responses to fluctuating CO concentrations were dynamically evaluated using the Coburn model prediction of the COHb resulting from one hour's exposure as the initial COHb level for the next hour. Twenty sets of 1-hour average CO concentrations patterns were evaluated to obtain the ranges of COHb shown for a given case and standard.

[c] Coburn model parameters: (All cases: ventilation rate = 10 liters/min)

Case 1: As given in Table 4.2-9, except a single ventilation rate used.

Case 2: Hemoglobin = 13 g/100ml; initial COHb = 0.7%; endogenous rate = 0.014 ml/min; blood volume = 3500 ml; CO lung diffusivity = 40 ml/min/torr; Haldane constant = 246.

Case 3: Same as for Case 2, except for difference in altitude.

Case 4: Hemoglobin = 9 g/100ml; initial COHb = 1.9%; endogenous rate = 0.03 ml/min; blood volume = 4600 ml; CO lung diffusivity = 25 ml/min/torr; Haldane constant = 218.

* Reproduced from Biller and Richmond (1982)

TABLE 4.2-11

SUMMARY OF ESTIMATED CRITICAL HEALTH EFFECT LEVELS FOR CARBON MONOXIDE (CO) EXPOSURE

		Approximate Ambient CO Levels (mg/m^3) to produce stated COHb in resting Individuals		
Effect	COHb %	1-hour	8-hour	References
Physiological Norm:	0.3-0.7	0	0	Coburn et al., 1969
Effects at Approximately:	2.5-3.0	79-97 (70-85ppm)	17-21 (15-18ppm)	
- Aggravation of cardiovascular diseases:				
Decreased exercise capacity in patients with angina pectoris* or peripheral arteriosclerosis				Anderson et al 1973; Aronow and Isbell, 1973; Aronow et al., 1979
*this effect may occur at levels as low as 2.0% or less				Aronow, 1981
Effects at Approximately:	3.0-6.5	97-239 (85-207ppm)	21-52 (18-45ppm)	
- Aggravation of cardiovascular disease:				
Changes in heart functioning; possible increase in cardiac death rate				Aronow et al., 1974;(see also Keeney et al., 1983;and Shephard, 1983)
- Behavioural changes:				
Impairment of vigilance tasks in healthy individuals**				
**this effect may possibly occur at lower levels				e.g. Beard and Wartheim, 1967

TABLE 4.2-11 (CONT'D)

Effect	COHb %	Approximate Ambient CO Levels (mg/m^3) to produce stated COHb in resting individuals		References
		1-hour	8-hour	
- Changes in work performance:				
Decreased exercise performance in healthy persons* and in patients with chronic lung disease *this effect may occur at lower levels				Aronow and Cassidy, 1975; Aronow *et al.*, 1977 eg. Drinkwater *et al.*, 1979
- Impaired fetal development:				
Resulting in subtle effects such as retardation of reading and mathematical ability in childhood* *if this is confirmed, maternal COHb of 4.1-6.7% may be responsible				see Shephard, 1983
Effects at Approximately:	5.0-20.0	176-887 (155-175ppm)	38-193 (33-170ppm)	
- Behavioural changes:				
Decrease in visual perception, manual dexterity, ability to learn, or perform complex sensorimotor tasks (such as driving)				eg. Bender *et al.*, 1971
- Changes in work performance:				
Decrease in maximum work time or oxygen consumption* *may be highly variable				eg. Ekblom and Huot, 1972; Horvath *et al.* 1975 Ayres *et al.*, 1969, 1970

4.2.3 Nitrogen Oxides

Introduction

The major effects of nitrogen oxides on human health relate to damage of respiratory tissue. Of these compounds, only nitrogen dioxide can be considered a health hazard at levels approximating those found in ambient air. There appears to be concern regarding the effects of both short-and long-term exposures (EPA, August 1982), although data with respect to long-term effects is sorely lacking.

The effect of NO_2 on airway resistance and other parameters of pulmonary function has been well documented in controlled human clinical studies, but these studies are limited to examining the effects of single, short term exposures, and, thus, do not directly address effects that may be caused by repeated exposures over long periods of time. Increased incidence of respiratory illness attributable to NO_2 exposure has indeed been suggested by a number of epidemiological studies, although methodological restrictions have generally cast doubt on the quantitative extrapolations of these findings. The ability of NO_2 to damage lung tissue and increase susceptibility to respiratory infection has been demonstrated by numerous animal toxicological studies. The nature of the deleterious effects attributable to NO_2 will be discussed below, along with the sort and quality of the substantiating evidence, and the consensus within the scientifc community as to exposure levels at which these effects may be expected to occur.

Nitrogen oxides, of course, also contribute indirectly to health effects through their role in the formation of ozone and acid rain. The adverse health effects attributable to these latter substances will be discussed in Section 4.2.4 and 4.2.7, respectively.

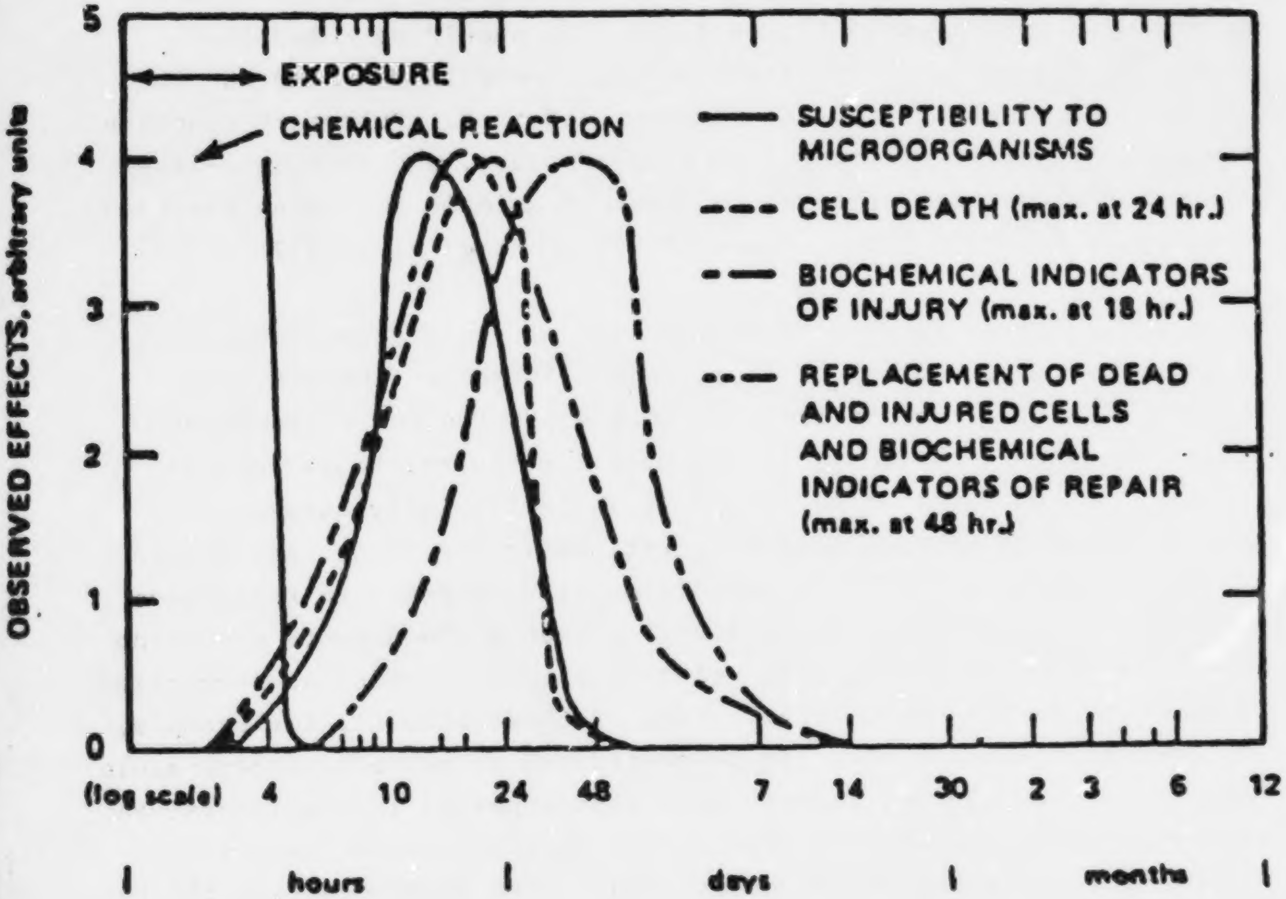
The Nature and Mechanism of NO_2 Toxicity: A Review of the Animal Data

The highly reactive but relatively insoluble nature of NO_2 results in chemical interaction and absorption along the entire tracheobronchial tree. The sequence of events that occurs in the respiratory system of animals following short term single exposures of 4 hours or less has been well described and is portrayed in Figure 4.2-6. It is important to note that the effects of NO_2 may not peak for several hours after exposure, and subjects may not fully recover for several days after removal from exposure.

The sequence of events during long-term continuous exposure of animals to NO_2 has been shown to be similar to the sequence for short-term exposures as portrayed in Figure 4.2-7. It should be noted that susceptibility to infection rises nearly linearly over time due to the increasing destruction of pulmonary defenses. Pulmonary function changes begin after a longer period of exposure, and emphysema-like alterations have been reported to occur in animals following extended exposure to relatively low levels of NO_2 (Port et al., 1977).

Table 4.2-12 presents some of the effects of short-term exposure to NO_2 observed in animal studies, as summarized by the U.S. EPA (August, 1982). It seems that multiple exposures in the range of 0.38 to 0.94 mg/m^3 (0.2 ppm to 0.5 ppm) for several hours account for many of the reported effects. It is particularly noteworthy that exposure of animals to concentrations in the range that occurs in ambient air appears to be associated with decrease in resistance to bacterial infection. Table 4.2-13 presents the effects occurring in animals exposed to NO_2 over relatively long periods, as summarized by the U.S. EPA (August, 1982). Some of these effects, for example, emphysematous alterations, can be considered as serious, irreversible health effects. Recent reports have also appeared in the literature implicating NO_2 exposure to effects not listed in these tables. For example, Richters and Richters (1983) have concluded from their animal experiments that NO_2 facilitates blood-borne cancer cell metastasis. Attempts are underway to quantitatively extrapolate the results of the animal studies directly to humans. In the interim, these studies serve at least to alert to potential serious health effects in humans.

Figure 4.2-6 Temporal Sequence of Injury and Repair Hypothesized From Short-Term Single Nitrogen Dioxide Exposures of Less Than 4 Hours*



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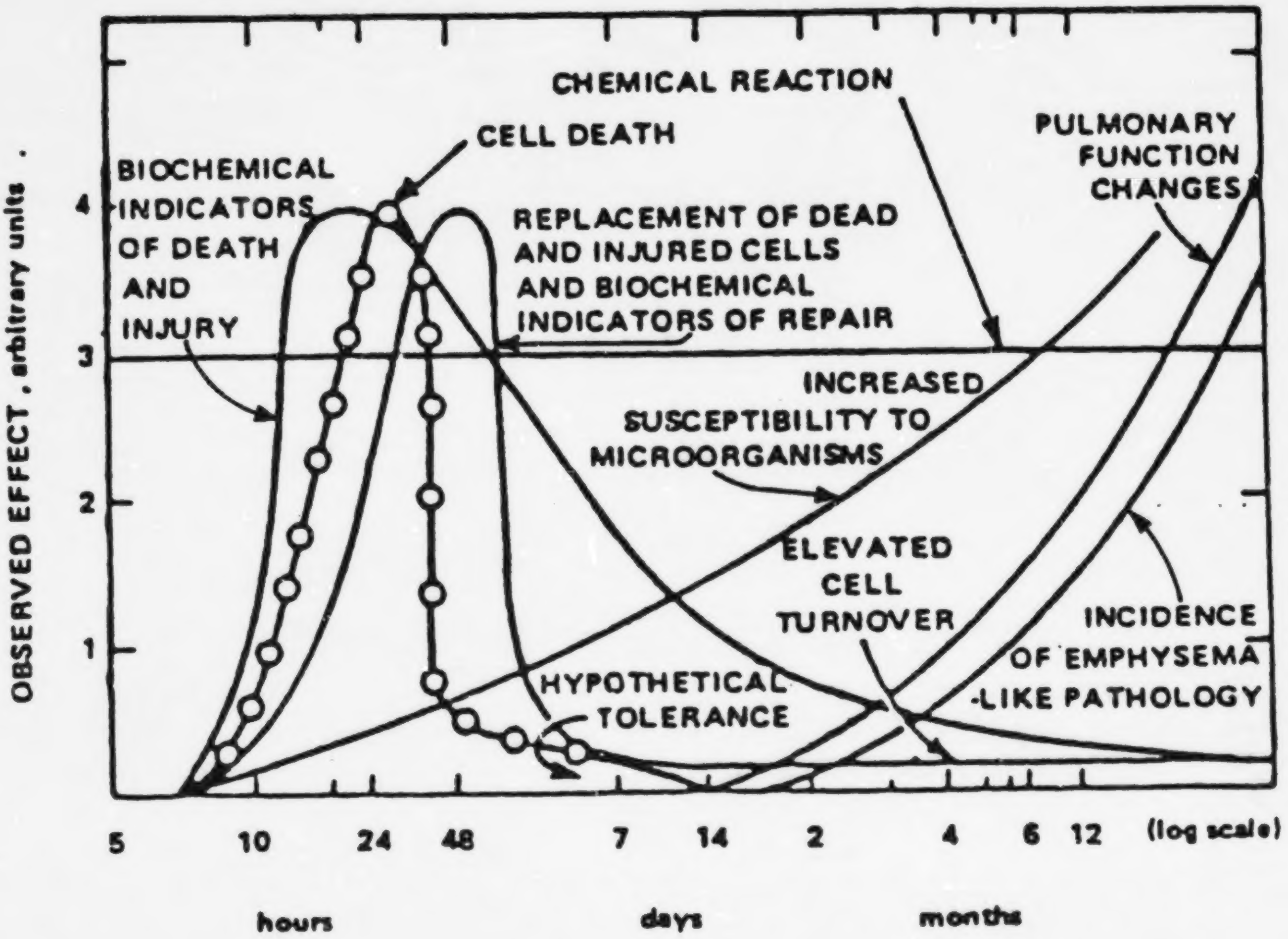
*Reproduced from E.P.A., Aug. 1982.

Figure 4.2-7 Temporal sequence of injury and repair hypothesized from continuous exposure to NO_2 as observed in experimental animals.* (4 on y-axis is equivalent to 100% of Observed Effects)



*Reproduced from E.P.A., Aug. 1982.

Table 4.2-12 Selected Animal Studies Demonstrating Effects for Short-Term Exposures to NO_2*

NO_2 Concentration $\mu g/m^3$	ppm	Duration of Exposure	Biological Effect	Implications of Effect	References
380 3760 35,500	0.20 2.0 19.0	3 hours	Inhibition of prostaglandin E_2 breakdown after 18-hour delay (rat)	Significant time delay before appearance of effect; interference of NO_2 with hormone metabolism	Menzel, 1980
380 to 94,000	0.20 to 50	4 hours	*In vivo* biosynthesis of nitrosomorpholine after pre-exposure to morpholine (mouse)	*In vivo* biosynthesis of carcinogenic compounds following exposure to NO_2	Iqbal et al., 1980
470	0.25	3 hours	Increased pentobarbital-induced sleep time in females; effect disappeared for repeated exposures (mouse)	Suggests NO_2 interference with xenobiotic (liver) metabolism. Females more sensitive to a single NO_2 exposure; adaptation or tolerance; extrapulmonary effect	Miller et al., 1980
470	0.25	4 hours/day, 5 days/week, 24 or 36 days	Isolated swollen collagen fibers (rabbits)	Repeated short-term exposures to NO_2 induce morphological alterations; extrapulmonary effect	Buell, 1970
750	0.40	4 hours/day, 7 to 14 days	Proteinuria (proteinuim urine); an analysis revealed presence of albumin and α, β, γ globulins (guinea pig)	Repeated short-term exposures to NO_2 induce kidney damage	Sherwin and Layfield, 1974
940	0.50	6, 18 or 24 hr/day, 12 months	Alveolar damage (mouse)	Repeated exposures to NO_2 induce morphological changes which reduce oxygen transfer capacity of lungs	Blair, et al., 1969
750	0.40	4 hours/day, 7 days	Acid phosphatase levels increased (guinea pig)	Repeated short-term exposures to NO_2 alter enzyme levels in the lungs	Sherwin et al., 1974
940	0.50	8 hours/day, 7 days	Increase in serum enzyme (LDH, CPK, SGOT, SGPT, CHE, lysozyme) levels in lungs; decrease in red blood cell glutathione peroxidase levels (guinea pig)	Repeated exposures to NO_2 alter enzyme levels in lungs, indicative of generalized damage to the lung	Donovan et al., 1976 Menzel et al., 1977
940	0.50	Intermittent 6 to 18 hr/day, for 6 months followed by challenge to *K. Pneumoniae*	18% increased mortality ($p < 0.05$) over controls due to decreased resistance to infection (mouse)	Repeated exposures to NO_2 reduce resistance to bacterial infections	Ehrlich and Henry, 1968
940 and 1880	0.50 and 1.0	4 hours and 1 hour	Degranulation of mast cells (rat)	Single NO_2 exposures cause release of substances with various activities including ability to increase airway resistance	Thomas et al., 1967
1000	0.53	8 hours/day 180 days	Alterations in levels of variety of brain enzymes (guinea pigs)	Repeated NO_2 exposures may induce changes in brain enzyme levels; extrapulmonary effect	Drodz et al., 1975
1880 3760 5600	1.0 3.0	3 hours followed by challenge with *S. pyogenes*	Increased mortality only for animals exposed to 3 ppm NO_2 during exercise (mouse)	Single exposure to NO_2 during exercise increase susceptibility to infection at or above 2.0 ppm	Gardner and Graham, 1976
2800	1.5	Continuous or 7 hr/day 7 days/wk followed by challenge with *S. pyogenes*	After 1 week, mortality with continuous exposure greater ($p < 0.05$) than that for intermittent. After 2 weeks, no significant difference between continuous and intermittent exposure (mouse)	Even though total dose is greater for continuous exposure, susceptibility to infection becomes equivalent for continuous and intermittent over time, suggesting repeated peaks are more important than continuous levels of NO_2	Illing et al., 1980
3760	2.0	3 hours followed by challenge with *S. pyogenes*	Increased mortality ($p < 0.05$) (mouse)	Single exposure to NO_2 can increase susceptibility to infection	Ehrlich et al., 1977
6600	$\leq$ 3.5	$\leq$ 6 hours followed by challenge with *S. pyogenes*	Increased mortality by 31.9%. Concentration (ppm) x time (hr) = 21 (mouse)	For a given dose (CXT) concentration has a greater effect than time	Gardner et al., 1979
6600	3.5	Continuous or 7 hours/day 7 days/week 15 days	Increased mortality with increased duration of exposure. No significant difference between continuous and intermittent exposure. With data adjusted for total difference in CxT mortality essentially the same (mouse)	Concentration is more important than duration of exposure in determining effects from a given dose	Gardner et al., 1979

*Reproduced from E.P.A., Aug. 1982.

See source for complete references

Table 4.2-13 Selected Animal Studies Demonstrating Effects for Long-Term Exposures to NO_2*

NO_2 Concentration mg/m^3	ppm	Duration of Exposure	Biological Effect	Implications of Effect	References
188	0.1	Continuous for 6 months with 1.0 ppm spikes for 2 hr/day	Structural alterations in bronchioles and alveolar ducts (mouse)	Combination of very low level continuous exposure to NO_2 with daily peaks causes emphysema like changes in relatively short time period	Port et al., 1977
680	0.36	Continuous for 7 days	Red blood cell D-2,3-diphosphoglycerate was significantly increased ($p < 0.05$) (guinea pig)	This may be indicative of tissue deoxygenation	Mersch et al., 1973
750	0.40, 1.0	Continuous for 7 days	Increase in lung protein content (Vitamin C deficient guinea pig) in one study, but another at the lowest concentration	Most likely due to plasma leakage which may be indicative of pulmonary edema and cell death; Vitamin C deficiency increases susceptibility	Sherwin and Carlson, 1973 Selgrade et al., 1981
750 to 1880	0.4 to 1.0	Continuous for 17 to 18 months	Reduction in growth rate and body weight; growth improved by dietary Vitamin E supplement (mouse)	NO_2 impairs metabolism and growth process	Csallany, 1975 Csallany and Ayaz, 1978
940	0.5	5 days/week for 3 or 6 weeks	Increased retention of protein in pulmonary air spaces (mouse)	Suggestive of pulmonary edema	Sherwin et al., 1977
940	0.5	Continuous for 14 days	Protein (Albumin and globulins) in urine (guinea pig)	Suggestive of kidney damage	Sherwin and Layfield, 1974
940	0.5	Continuous for 7 days	Higher lysozyme, plasma cholinesterase, and other enzyme levels (guinea pig)	Indicative of liver and heart damage	Menzel et al., 1977 Donovan et al., 1976
940	0.5	Continuous for 4 months	Alterations in blood enzyme levels (guinea pig)	Indicative of liver damage (hepatic lesions)	Menzel et al., 1977 Donovan et al., 1976
940	0.5	Continuous for 90 days or 12 months followed by challenge of K. pneumoniae	Increased mortality ($p < 0.05$) after 90 days and after 12 months due to respiratory infection (mouse)	NO_2 increases susceptibility to respiratory infection	Ehrlich and Henry, 1968
940 to 1880	0.5 to 1.0	Continuous for 39 days followed by challenge of A/PR/8 virus	Significantly increased rate of respiratory infection (female mouse)	NO_2 increases susceptibility to respiratory infection	Ito, 1971
940	0.5	Continuous exposure for 5 days/week (21, 28, 33 weeks) with daily 1-hour peaks of 2 ppm	Alveolar macrophage damage and morphological alterations (mouse)	NO_2 reduced effectiveness of pulmonary defenses	Aranyi et al., 1976
940	0.5	Continuous for 12 months	Loss of cilia, alveolar edema, bronchial hyperplasia, fibrosis (mouse)	Various pulmonary effects indicative of potentially serious lung damage. Reduction in resistance to respiratory infection. Evidence suggestive of changes in terminal bronchioles; decreased clearance of particles	Hattori and Takemura, 1974 Hattori, 1973
940 to 1500	0.5 to 0.8	Continuous for 1 month	Damage to tracheal mucosa and cilia (mouse)	Reduction in resistance to respiratory infection; decreased clearance of particles	Hattori et al., 1972 Nakajima et al., 1969
1500	0.8	Continuous for 33 months	Decreased respiratory rate (~ 20%). Gross and microscopic alterations (rats)	Evidence suggestive of microscopic changes in terminal bronchioles	Freeman et al., 1966 170
1880	1.0	Continuous for 493 days, challenge 5 times with monkey adapted influenza virus	Reduced immunological activity; slight emphysema thickened bronchial and bronchiolar epithelium (monkey)	NO_2 reduces effectiveness of pulmonary defenses and may induce morphological changes	Fenters et al., 1966
1880	1.0	6 months continuous, followed by intranasal challenge with D. pneumoniae	Increased respiratory infection and mortality (guinea pig)	NO_2 reduces ability to defend against pulmonary infection	Kosmider et al., 1973
3760	2.0	Continuous for 43 days	No changes in terminal bronchi. Cilia lost and altered by 72 hours. Greater cilia loss and focal hyperplasia by 7 days. Regeneration of cilia by 14 days. Substantial recovery by 21 days (rat)	Suggestive of adaptation or tolerance to NO_2	Stephens et al., 1972
3760	2.0	Continuous for 14 months	Hypertrophic epithelium, particularly in the area of respiratory bronchiole (monkey)	Advanced stage of morphological damage	Furiosi et al., 1973

* Reproduced from E.P.A., Aug. 1982 See source for complete references

Health Effects Attributable to Single or Short-Term Exposures: Results of Experimental Studies in Humans

Table 4.2-14 summarizes the major effects of NO_2 exposure reported from clinical human studies (EPA, September 1982). Similar summaries have been prepared by the WHO (1977), Burton et al. (1981) and Ferris(1982). Each of these health effects will be elaborated upon briefly below.

Pulmonary Function Changes: It has been clearly shown that increased airway resistance and other changes indicative of impaired pulmonary function occur in healthy adults exposed to single 2-hour NO_2 concentrations ranging from 4.7 to 13.2 mg/m^3 (2.5 to 7.0 ppm) (Yokoyama 1972, Nieding et al. 1970, 1973 and 1977; Abe 1967, Beil and Ulmer 1976). Certain studies also indicate statistically significant pulmonary effects in healthy subjects after shorter duration exposures (3-15 min.) to NO_2 concentrations below 3.8 mg/m^3 (2.0 ppm) alone or in combination with NaCl aerosol (Nakamura 1964; Abe 1967; Nieding et al. 1970, 1973; Suzuki and Ishikawa 1965). One study (Suzuki and Ishikawa 1965) reported altered respiratory function after exposure to NO_2 levels of 1.3-3.8 mg/m^3 (0.7 - 2.0 ppm) for 10 minutes, although it has been pointed out (EPA, September 1982) that a clear association between the observed effects and any particular concentration within this range was not possible. The EPA also noted that Hackney et al. (1978) found only a marginal loss in Forced Vital Capacity (FVC; the amount of air that can be expired from the lungs after a maximal inspiration) after a 2 hour exposure to 1.9 mg/m^3 (1.0 ppm) NO_2 on two successive days. They therefore questioned the significance of this finding (ibid). Similarly, Kerr et al. (1979) reported changes in lung compliance in 10 healthy adults exposed for 2 hours to 0.94 mg/m^3 (0.5 ppm), but again the authors questioned the biological significance of their own findings and concluded that the exposure was not associated with significant decrement in pulmonary function. The conclusions reached by Beil and Ulmer (1976) and Folinsbee et al. (1978) that there was no physiological significant pulmonary effects at exposure levels of 1.88 and 1.13 mg/m^3 (1.0 and 0.6 ppm) NO_2 for 2

Table 4.2-14 EFFECTS OF EXPOSURE TO NITROGEN DIOXIDE ON PULMONARY FUNCTION IN CONTROLLED STUDIES OF HEALTHY HUMAN ADULTS**,***

Concentration		Pollutant	No. of Healthy Subjects	Exposure Time	Effects	Reference
µg/m³	ppm					
13,000	7.0	NO_2	Several	10-120 min.	Increased R_{aw}* in some subjects. Others tolerated 30,000 µg/m³ (16 ppm) with no increase in R_{aw}.	Yokuyama, 1972
9,400	5.0	NO_2	11	2 hrs.	Increase in R_{aw}* and a decrease in $AaDO_2$* with intermittent light exercise. No$_3$ enhancement of the effect when 200 µg/m³ (0.1 ppm) O_3 and 13,000 µ/m³ (5.0 ppm) SO_2 were combined with NO_2 but recovery time apparently extended.	von Nieding et al., 1977
9,400	5.0	NO_2	16	15 min.	Significant decrease in DL_{CO}*	von Nieding et al., 1973
9,400	5.0	NO_2	13	15 min.	Significant decrease in PaO_2* but end expiratory PO_2* unchanged with significant increase in systolic pressure in the pulmonary artery	von Nieding et al., 1970
7,500 to 9,400	4.0 to 5.0	NO_2	5	10 min.	40% decrease in lung compliance 30 min. after exposure and increase in expiratory and inspiratory flow resistance that reached maximum 30 min. after exposure.	Abe, 1967
5,600	3.0	NO2	1	5 min.	Increase in R_{aw}* compared to pre-exposure values (enhanced by NaCl aerosol).	Nakamura, 1964
11,300	6.0	NO_2	1	5 min.	More subjects were tested at higher exposures.	
1,880	1.0	NO_2	8	2 hrs.	No increase in R_{aw}.	Beil and Ulmer, 1976
4,700	2.5	NO_2	8	2 hrs.	Increased R_{aw} with no further impairment at higher concentrations. No change in arterial PO_2 pressure or PCO_2 pressure.	
14,000	7.5	NO_2	16	2 hrs.	Increased sensitivity to a bronchoconstrictor (acetylcholine) at this concentration but not at lower concentrations.	
9,400	5.0	NO_2	8	14 hrs.	Increase in R_{aw} during first 30 min. that was reduced through second hour followed by greater increases measured at 6, 8 and 14 hrs. Also increased susceptibility to a bronchoconstrictor (acetylcholine).	
1,300 to 3,800	0.7 to 2.0	NO_2	10	10 mins.	Increased inspiratory and expiratory flow resistance of approximately 50% and 10% of control values measured 10 mins. after exposure.	Suzuki and Ishikawa, 1965
1,880	1.0	NO_2	16	2 hrs.	No statistically significant changes in pulmonary function tests with exception of small changes in FVC. (See page 1-9 and 15-17).)	Hackney et al. 1978
1,150	0.6	NO_2	15	2 hrs.	No physiologically significant changes in cardiovascular, metabolic, or pulmonary functions after 15, 30 or 60 mins. of exercise.	Folinsbee et al., 1978

*** Reproduced from E.P.A. Sept. 1982; See source for complete references

(Cont'd)

Table 4.2-14 (Continued)

Concentration		Pollutant	No. of Healthy Subjects	Exposure Time	Effects	Reference
µg/m³	ppm					
1,000	0.50	O_3	4	4 hrs.	With each group minimal alterations in pulmonary function caused by O_3 exposure. Effects were not increased by addition of NO_2 or NO_2 and CO to test atmospheres.	Hackney et al., 1975
1,000 with	0.50	O_3				
560	0.29	NO_2				
1,000 with	0.50	O_3				
560 and	0.29	NO_2				
45,000	30.0	CO				
500	0.25	O_3	7	2 hrs.	Little or no change in pulmonary function found with O_3 alone. Addition of NO_2 or of NO_2 and CO did not noticeably increase the effect. Seven subjects included some believed to be unusually reactive to respiratory irritants.	Hackney et al., 1975
500 with	0.25	O_3				
560	0.29	NO_2				
500 with	0.25	O_3				
560 and	0.29	NO_2				
45,000	30.0	CO				
1,880 to 3,760	1.0 to 2.0	NO_2	10	2 1/2 hrs	Alternating exercise and rest produced significant decrease for hemoglobin, hematocrit, and erythrocyte acetylcholinesterase.	Posin et al., 1978
100 with	0.05	NO_2	11	2 hrs.	No effect on R_{aw} or $AaDO_2$; exposed subjects showed increased sensitivity of bronchial tree to a bronchoconstrictor (acetylcholine) over controls not exposed to pollutants.	von Nieding et al., 1977
50 and	0.025	O_3				
300	0.11	SO_2				

*R_{aw} : airway resistance

$AaDO_2$: difference between alveolar and arterial blood partial pressure of oxygen

DL_{CO} : diffusion capacity of the lung for carbon monoxide

PaO_2 : arterial partial pressure of oxygen

PO_2 : partial pressure of oxygen

PCO_2 : partial pressure of carbon dioxide

**By descending order of lowest concentration evoking a significant effect.

*** Reproduced from E.P.A. Sept. 1982; See source for complete references

(Cont'd)

Table 4.2-14 (continued)

EFFECTS OF EXPOSURE TO NITROGEN DIOXIDE ON SENSORY RECEPTORS IN CONTROLLED HUMAN STUDIES ***

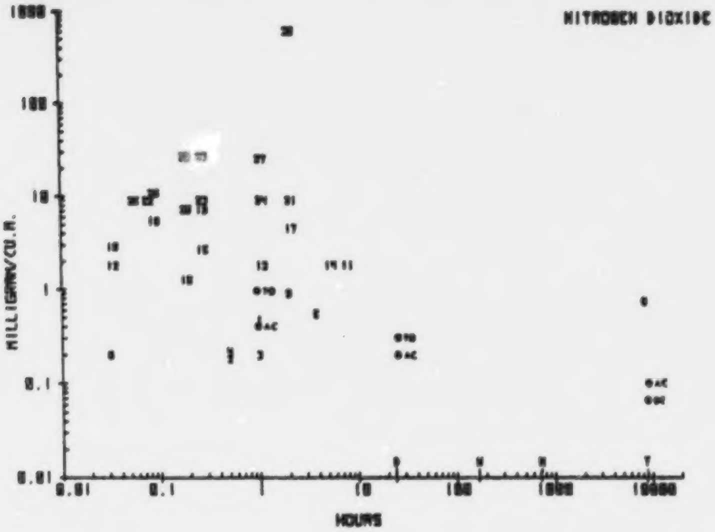
NO_2 Concentrations µg/m³	NO_2 Concentrations ppm	No. of Subjects	Time until effect	Effects	No. of Subjects Responding	Reference
790	0.42	8	Immediate	Perception of odor of NO_2	8/8	Henschler et al., 1960
410	0.22	13	Immediate	Perception of odor of NO2	8/13	*Ibid*.
230	0.12	9	Immediate	Perception of odor of NO_2	3/9	*Ibid*.
230	0.12	14	Immediate	Perception of odor of NO_2	Most	Shalamberidze,
200	0.11	28	Immediate	Perception of odor of NO_2	26/28	Feldman, 1974
0 to 51,000	0 to 27	6	54 minutes	No perception of odor of NO_2 when concentration was raised slowly from 0 to 51,000 µg/m³	0/6	Henschler et al., 1960
2,260	1.2	6	Immediate	Perception of odor improved when relative humidity was increased from 55% to 78%	6/6	*Ibid*.
140	0.07	4	5 and 25 minutes	Impairment of dark adaptation	4/4	Shalamberidze,
150 to 500	0.08 to 0.26	5	Initial	Increased time for dark adaptation at 500 µg/m³ (0.26 ppm)	Not Reported	Bondareva, 1963
			Repeated over 3 months	Initial effect reversed		

*** Reproduced from EPA September 1982; See source for complete references hours have also been noted by the U.S. regulatory authorities (EPA, August 1982) as were the conclusions reached by Hackney et al. (1975a, b and c) and Von Nieding et al. (1977) that there were not physiological significant effects at NO_2 levels below 0.56 mg/m^3 (0.3 ppm) in the presence of various other air pollutants.

Burton et al. (1981) displayed the 'effect' and 'no effect' studies graphically to aid the Canadian authorities in their review of the air quality criteria for NO_2, as shown in Figure 4.2-8 and Table 4.2-15, and Figure 4.2.9 and Table 4.2-16, respectively. These figures illustrate that there is considerable divergence among the studies and that there are some investigators who have reported effects at levels at or below the current tolerable levels, and some who failed to find effects at concentrations even above these levels. This endeavour also indicates which studies most merit careful evaluation.

The EPA noted that exception reported by Nieding et al. (1977) that increased sensitivity to bronchoconstriction occurred at 0.094 mg/m^3 (0.05 ppm) NO_2 in the presence of 0.05 mg/m^3 (0.025 ppm) ozone and 0.29 mg/m^3 (0.11 ppm) SO_2. However, the EPA felt that in view of 1) the controversy over the health significance of altered sensitivity to bronchoconstriction, 2) uncertainty due to methodological differences between his techniques and those used by other investigators, and 3) lack of confirmation of these results by other researchers, this finding cannot be regarded as conclusive evidence for respiratory effects occurring at NO_2 concentrations substantially below 1.88 mg/m^3 (1.0 ppm) for healthy adult subjects. In their conclusions, the EPA took into account the health significance of the alterations noted by pointing out that concentrations above 9.4 mg/m^3 (5.0 ppm) for as little as 15 minutes would increase airways resistance in healthy human adults, as well as impair normal gas transport, however, concentrations of 4.7 mg/m^3 (2.5 ppm) for 2 hours would increase airways resistance but would not impair gas transport, as would single exposures for 15 minutes to NO_2 at concentrations of 3.0 mg/m^3 (1.6 ppm). They felt that single exposures for times ranging from 15 minutes to 2 hours

Figure 4.2-8 Relevant Studies Demonstrating Effects of Nitrogen Dioxide on Humans Shown According to Concentration and Duration of Exposure*



CODES:

TO = Canada's existing tolerable level of NO_2
AC = Canada's existing acceptable level of NO_2
DE = Canada's existing desirable level of NO_2
AH = Animal: Human; AL = effect
* Reproduced from Burton et al., 1981. See source for complete references.

Table 4.2-15 Data List for Nitrogen Dioxide Effects on Humans*

POLLUTANT : NITROGEN DIOXIDE

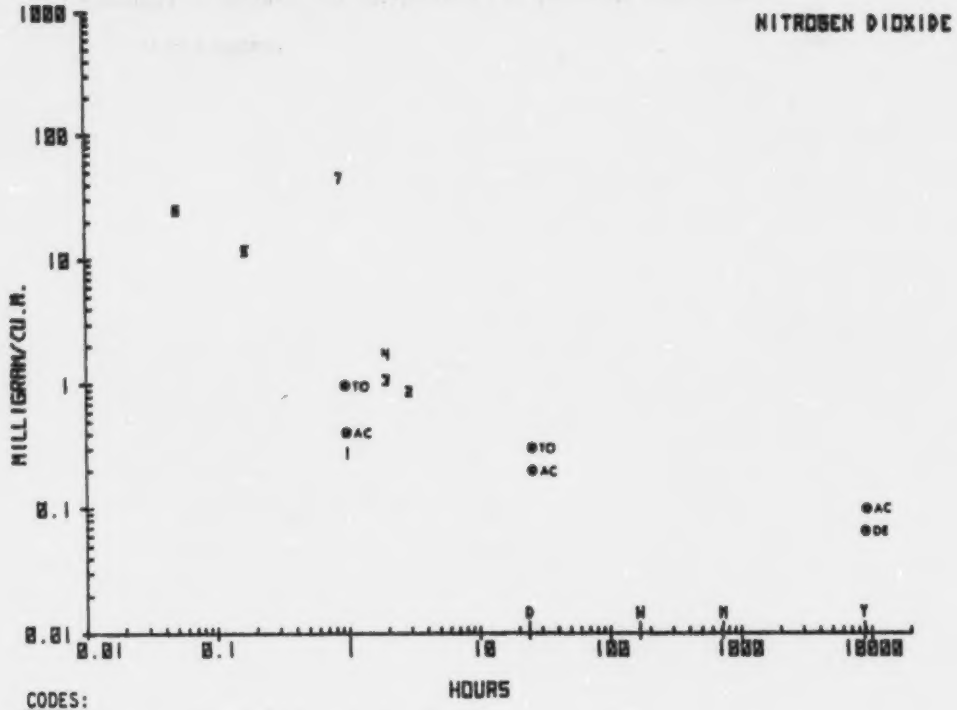
CONCENTRATION MG/M↑3	PPM	TIME HOURS	EXPOSURE TYPE	#	REFERENCE	CATEG
● 0.49	0.26	1.00	C	1	BONDAREVA 1963	AHO
● 0.19	0.10	0.50	C	2	VOISIN ET AL 1977	AHO
● 0.21	0.11	1.00	C	3	OREHEK ET AL 1976	AHO
● 0.23	0.12	0.50	C	4	HENSCHLER ET AL 1960	AHO
● 0.56	0.30	3.00	C	5	CASE ET AL 1975	AHO
0.75	0.40	8760.00	I	6	KOSMIDEP & MISIEWICZ 1973A	AHO
0.75	0.40	8760.00	I	7	KOSMIDER & MISIEWICZ 1973B	AHO
● 0.21	0.11	0.03	C	8	FELDMAN 1974	AHO
● 0.94	0.50	2.00	C	9	KERR ET AL 1978	AHO
● 1.32	0.70	0.17	C	10	SUZUKI & ISHIKAWA 1965	AHO
1.88	1.00	7.50	I	11	BUCKLEY ET AL 1977	AHO
● 1.88	1.00	0.03	C	12	SMIDT & VON NIEDING 1974	AHO
1.88	1.00	1.00	C	13	VON NIEDING & WAGNER 1975	AHO
1.88	1.00	5.00	I	14	POSIN ET AL 1978	AHO
2.82	1.50	0.25	C	15	VON NIEDING ET AL 1973	AHO
● 3.01	1.60	0.03	C	16	VON NIEDING ET AL 1971	AHO
4.70	2.50	2.00	C	17	BEIL & ULMER 1976	AHO
5.64	3.00	0.08	C	18	NAKAMURA 1964	AHO
7.52	4.00	0.25	C	19	VON NIEDING ET AL 1971	AHO
7.52	4.00	0.17	C	20	ABE 1967	AHO
9.40	5.00	2.00	C	21	VON NIEDING & WAGNER 1977	AHO
9.40	5.00	0.07	C	22	STRESEMANN & VON NIEDING 1970	AHO
9.40	5.00	0.25	C	23	VON NIEDING ET AL 1973	AHO
9.40	5.00	1.00	C	24	YOKOYAMA 1967B	AHO
9.40	5.00	0.05	C	25	VON NIEDING & KREKELER 1971	AHO
11.28	6.00	0.08	C	26	NAKAMURA 1964	AHO
26.32	14.00	1.00	C	27	YOKOYAMA 1967B	AHO
28.20	15.00	0.25	C	28	VON NIEDING & WAGNER 1975	AHO
28.20	15.00	0.17	C	29	YOKOYAMA 1970	AHO
632.81	336.50	2.00	C	30	CASSAN ET AL 1976	AHO

CODES:

● Papers considered critical to the revision of the existing subjectives
Exposure Type: C = constant; I - intermittent
= refer to Figure 4.2-8
Category: A = Animal, H = Human; O = effect

*Reproduced from Burton et al., 1981. See source for complete references.

Figure 4.2-9 Relevant Studies Demonstrating No Effects of Nitrogen Dioxide on Humans Shown According to Concentration and Duration of Exposure*



CODES:

TO = Canada's existing tolerable level of NO_2

AC = Canada's existing acceptable level of NO_2

DE = Canada's existing desirable level of NO_2

AH = Animal: Human; AL = effect

* Reprodu:ed from Burton *et al.*, 1981. See source for complete references.

Table 4.2-16 Data List for No Effects of Nitrogen Dioxide on Humans*

POLLUTANT : NITROGEN DIOXIDE

CONCENTRATION MG M↑3	PPM	TIME HOURS	EXPOSURE TYPE	#	REFERENCE	CATEGORY
● 0.30	0.16	1.00	C	1	BONDAREVA 1963	AH1:
● 0.94	0.50	3.00	C	2	HACKNEY ET AL 1973	AH:
● 1.17	0.62	2.00	C	3	FOLINSBEE ET AL 1978	AH:.
● 1.88	1.00	2.00	C	4	HACKNEY ET AL 1978	AH1:
● 13.16	7.00	0.17	C	5	YOKOYAMA 1967A	AH1.
● 28.20	15.00	0.05	C	6	DE VRIES 1976	AH:
● 50.76	27.00	0.90	C	7	HENSCHLER ET AL 1960	AH.1

CODES:

Papers considered critical to the revision of the existing subjectives

Exposure Type: C = constant; I = intermittent

# = refer to Figure 4.2-8

Category: A = Animal; H = Human; O = effect

* Reproduced from Burton et al., 1981. See source for complete references.

to NO_2 at concentrations of 2.8 mg/m³ (1.5 ppm) or below have not been shown to affect respiratory function in healthy individuals (or for that matter, in bronchitics).

Symptomatic Effects: The EPA (August 1982) dissected out the studies reporting symptomatic effects in individuals experimentally exposed to NO_2 in an attempt to better appraise the health significance of the observed pulmonary function changes. Hackney et al., (1978) observed an increase in symptomatic effects (cough, chest tightness, laryngitis and nasal discharge) in 5 of 16 healthy adults exposed to 1.88 mg/m³ (1.0 ppm) NO_2 for 2 hours. The EPA (ibid) noted, however, that the difference failed to reach statistical significance and that the health significance of the symptom increases was questionable. In the study by Kerr et al., (1979), only 1 of 10 healthy subjects reported mild symptomatic effects associated with exposure to 0.94 mg/m³ (0.5 ppm) NO_2 for two hours, however, 7 of 13 asthmatics reported various symptoms during or after exposure to 0.94 mg/m³ (0.5 ppm) NO_2 for 2 hours, with 15 minutes of light or moderate exercise during the exposure. The particular relevance to NO_2 exposure on individuals with chronic lung disease will be summarized below.

Non-Pulmonary Effects: Detection of NO_2 as a noxious odour has been shown to occur at concentrations as low as 0.21 mg/m³ (0.11 ppm) (Feldman 1974). Effects on sensory perception functions, such as dark adaptation, occur in human subjects at NO_2 exposures as low as 0.13 to 0.15 mg/m³ (0.07 to 0.08 ppm) (Shalamberidze 1967; Bondareva 1963).

Health Effects of Long Term Continuous Exposure and/or Repeated Short Term Peaks: Results of Epidemiological Studies.

It is generally believed that the most serious health effects of NO_2 are probably attributable to repeated exposures to peak concentrations rather than single exposures at continuous low level concentrations

(EPA, August 1982; WHO 1977). These effects cannot be examined in controlled human experiments for obvious ethical reasons, and reliance on epidemiological studies therefore becomes necessary. Unfortunately, as explained in Section 4.2.1, interpretation of epidemiological studies, or the effects of individual pollutants, is unavoidably complicated by the complex mixture of pollutants in ambient air. Moreover, community studies on the effects of NO_2 exposure conducted prior to 1973 are of particularly questionable validity due to the use of the Jacobs-Hocheiser method of measuring atmospheric concentrations of NO_2.

Community Studies: Among these studies were those conducted by Shy et al. (1970a and b) and Pearlman et al. (1971) in Chattanooga which housed a TNT plant, providing a large source of ambient NO_2. These investigations reported small, but statistically significant decreases in Forced Expiratory Volume (FEV: the amount of air that can be exhaled in a given time period, after maximal inspiration) in 7 to 8 year old children living in areas with higher NO_2 concentrations, compared to those living in lower NO_2 areas. They also reported an increased incidence of acute respiratory disease in high NO_2 areas. The EPA (August 1982), the WHO (1977) and others (eg. Roth et al. 1982; Ferris, 1982) have questioned the physiological and statistical significance of these findings, as well as the NO_2 exposure estimates to which the reported effects have been attributed.

The results of these and other community epidemiology studies are summarized in Table 4.2-17 (see Burton et al. 1981 for a more comprehensive list and detailed description of each study). Most of the studies indicated that no significant pulmonary function effects were observed in populations exposed to peak levels of NO_2, alone or in combination with other pollutants (all less than 1.88 mg/m^3 NO_2 (1.0 ppm) (Cohen et al., 1972; Speizer and Ferris 1972a and b; Burgess et al., 1973; Linn et al., 1976). The one exception was a study (Kagawa and Toyama 1975) which reported correlations between

TABLE 4.2-17

EFFECTS OF EXPOSURE TO NO_2 ON PULMONARY FUNCTION IN COMMUNITY EPIDEMIOLOGY STUDIES

NO_2 Exposure Concentrations		Other Pollutants			
μg/m^3	ppm	μg/m	Study Population	Reported Effects	References
Daily Mean Conc. 130-282	0.08-0.15	Sulphates: 10-13 SO_2: <26 Particulates: 63-96	School children in 5 areas of Chattanooga (795 2nd graders and their families)	Significant difference between results of high NO_2 areas and two companion areas, but no dose-response gradient. Increased frequency of respiratory illness in families in high NO_2 areas, but also no gradient demonstrated	Shy *et al.*, 1970a,b
Daily Mean Conc. 150-282	0.08-0.15	Sulphates: 10-13 SO_2: <26 Particulates: 63-96	1st and 2nd grade children (1906) and all Caucasian infants born during 1966-1968 (1311)	Increase in frequency of bronchitics (but not pneumonia or croup) in school children living in high NO_2 areas 1 or 2 years or more. No such association for infants	Pearlman *et al.*, 1971
Median hourly conc. 66-132	0.035-0.07	SO_2: 157 Particulates: 135 Oxidant: 137	205 office workers in L.A. 439 office workers in San Francisco	No differences in most tests. Smokers in both cities showed greater changes in pulmonary function than non-smokers	Linn *et al.*, 1976
Mean annual 24 h Conc. 100	0.055	SO_2: 91			
1 h mean High exposures: 260-560	0.14-0.30		128 traffic policeman in urban Boston and 140 patrol officers in nearby suburbs	No difference in various	Speizer and Ferris, 1973 Burgess *et al.* 1973
Low exposures: 110-170	0.06-0.09				
High exposure group: Estimated 1 h max. 480-960	0.26-0.51		Non-smokers in L.A. (adult)	No difference found in several ventilatory measurements including spirometry and flow volume curves	Cohen *et al.*, 1972
Annual mean 24 h conc: 96	0.051				
Low exposure group: Estimated 1 h max. 205-430	0.12-0.23				
Annual mean 24 h conc: 43	0.01				
1 h conc: 40-360	0.02-0.19		20 school age children 11 years of age	During warmer part of year, NO_2, SO_2 and TSP significantly correlated with V_{max} at 25% and 50% FVC specific airway conductance. Significant correlation between each of four pollutants (NO_2, NO, SO_2 and TSP) and V_{max} at 25% and 50% FVC; but no clear delineation of specific pollutant concentrations at which effects occur	Kagawa and Toyama, 1975

decrements in maximum expiratory flow rates or specific airway conductance and concentration of NO_2 and other pollutants in 20 Japanese school children. The 1-hour concentration during testing ranged from 0.04 to 0.36 mg/m^3 (0.02 to 0.19 ppm), but the data were felt (EPA, September 1982) to be such that no specific NO_2 levels within this range could be associated with the decrements noted.

In their overall assessment of the data, the EPA (August 1982) wrote "At best we can conclude that the findings of Shy _et_ _al_. (1970a,b, 1973), Pearlman _et_ _al_. (1971) and Kagawa and Toyama (1975) are not inconsistent with the hypothesis that NO_2, in a complex mix with other pollutants in the ambient air, adversely affects respiratory function and illness in children". They specifically noted that the community epidemiology studies did not take into account exposure to NO_2 generated by the use of gas stoves, which they felt had considerable relevance.

Studies Involving Homes with Gas Stoves: Table 4.2-18 summarizes the principal studies investigating effects of exposure to NO_2 in gas stove homes. Both British and U.S. studies on indoor pollution effects showed that children are at special risk for NO_2 induced acute respiratory disease (Mitchell _et_ _al_. 1974; Keller _et_ _al_. 1979a and b; EPA 1976). Several studies (Melia _et_ _al_. 1979; Goldstein _et_ _al_. 1979; and Florey _et_ _al_. 1979) found a weak association between gas cooking (shown to be associated with increased NO_2 levels) and respiratory illness in some groups of children. The EPA (September 1982) noted, however, that although findings in this area were conflicting, initial results from a continuing prospective epidemiological study revealed that even after correcting for potential confounding factors, a clear association existed between increased respiratory illness below age 2, and the presence of gas cooking devices; small

Table 4.2-18 COMPILATION OF REPORTED EFFECTS ASSOCIATED WITH EXPOSURE TO NO_2 IN THE HOME IN COMMUNITY STUDIES INVOLVING GAS STOVES[a]

μg/m³ Conversions	NO_2 Concentration (ppm)	Study Population	Reported Effects[b]	References
17.6-112.8 18.8- 94.0 75.2-112.8 1880	95th percentile of 24 hr avg in activity room 0.02 - 0.06 (gas) 0.01 - 0.05 (elec.) Frequent peaks in 1 home of 0.4-0.6 (gas). Maximum peak 1.0 (gas).	8,120 children, ages 6-10, 6 different cities, data also collected on history of illness before age 2	Significant association between history of serious respiratory illness before age 2 and use of gas stoves ($p < .01$). Also, small but statistically significant decreases in pulmonary function (FEV_1 and FVC) in children from gas stove homes.	Speizer et al., 1980
	NO_2 concentrations not measured at time of study.	2,554 children from homes using gas to cook compared to 3,204 children from homes using electricity, ages 6-11	Proportion of children with one or more respiratory symptoms or disease (bronchitis, day or night cough, morning cough, cold going to chest, wheeze, asthma) increased in homes with gas stoves vs. electric stove homes (for girls $p \simeq 0.10$; boys not sig.) after controlling for confounding factors.	Melia et al., 1977
	NO_2 concentrations not measured in some homes studied for health effects.	4827 children, ages 5-10	Higher incidence of respiratory symptoms and disease associated with gas stoves (for boys $p \sim 0.02$: girls $p \simeq 0.15$) for residences in urban but not rural areas, after controlling for confounding factors.	Melia et al., 1979
9.4-595.0 11.3-353.4 7.5-317.7 5.6- 69.6	Kitchens (weekly avg.): 0.005-0.317 (gas) 0.006-0.188 (elec.) Bedrooms (weekly avg.): 0.004-0.169 (gas) 0.003-0.037 (elec.)	808 children, ages 6-7	Higher incidence of respiratory illness in gas-stove homes ($p \simeq 0.10$). Prevalence not related to kitchen NO_2 levels, but increased with NO_2 levels in bedrooms of children in gas-stove homes. Lung function not related to NO_2 levels in kitchen or bedroom.	Florey et al., 1979 and Goldstein et al., 1979 (both are companion papers to Melia et al., 1979)
9.4-206.8 0-112.8 28.2- 94.0	Sample of households 24 hr. avg: 0.005-0.11 (gas) 0-0.06 (elec.) 0.015-0.05 (outdoors)	128 children, ages 0-5 346 children, ages 6-10 421 children, ages 11-15	No significant difference in reported respiratory illness between homes with gas and electric stoves in children from birth to 12 years.	Mitchell et al., 1974 See also Keller et al., 1979
	Sample of household same as reported above	174 children under 12	No evidence that cooking mode is associated with the incidence of acute respiratory illness.	Keller et al., 1979
	See above for monitoring.	Housewives cooking with gas stoves, compared to those cooking with electric stoves. 146 households.	No evidence that cooking with gas associated with an increase in respiratory disease.	Keller et al., 1979
	See above for monitoring.	Members of 441 households	No significant difference in reported respiratory illness among adults in gas vs electric cooking homes.	Mitchell et al., 1974 See also Keller et al., 1979a.
470-940 1880	Preliminary measurements peak hourly .25-0.50, max. 1.0	Housewives cooking with gas stoves, compared to those cooking with electric stoves	No increased respiratory illness associated with gas stove usage.	U.S. EPA, 1976

[a] Exposures in gas stove homes were to NO_2 plus other gas combustion products.

[b] Effects reported in published references are summarized here. However, the Criteria Document warns that considerable caution should be used in drawing firm conclusions from these studies.

*Reproduced from E.P.A., Aug. 1982, see source for complete references decrements in pulmonary function were also noted in school aged children (Speizer et al. 1980). Based on the cumulative findings from animal and human clinical studies, the EPA (August 1982) felt that NO_2 seemed indeed to be the principal agent responsible for the effects demonstrated in the gas stove studies. The fact that animal evidence has demonstrated that NO_2 impairs respiratory defense mechanisms provides a plausible basis for inferring that NO_2 may be associated with the reported increased incidence of acute respiratory illness in children living in homes with gas stoves.

Animal infectivity model studies (e.g. Gardner et al., 1977, 1979; Coffin et al., 1977) have suggested that brief exposures to high concentrations of NO_2 resulted in more severe infections and greater mortality than did prolonged exposures to lower concentrations. These findings not only further supported the hypothesis that NO_2 was indeed the causative agent, but suggested that peak exposures may be more important than long term low level exposures in causing the effects observed in the gas stove homes. Speizer et al. (1980) also felt that peak values were most important, based largely on the fact that long term (24 hours or longer) concentrations of NO_2 do not differ much in gas stove homes compared to electric stove homes. The EPA therefore hypothesized that the effects observed in the gas stove studies were likely attributable to repeated short term peak NO_2 exposures ranging up to 0.94-1.88 mg/m³ (0.5-1.0 ppm) rather than the annual average levels of NO_2 0.02-0.11 mg/m³ (0.01-0.06 ppm) which were observed (Spengler et al., 1979; Speizer et al., 1980).

Adverse Health Effects and Particularly Sensitive Populations

A judgement that must be made with regard to the above evidence relates to the degree of change in pulmonary function that should be considered an adverse health effect. As there is a large reserve capacity in the human respiratory system, even fairly large changes in pulmonary function may sometimes not be perceived by healthy adults. However, individuals with respiratory disease may already be functioning at or near the limit of their lung capacity, especially when engaged in light or moderate activity. A relatively small impairment of lung function in these people may affect their ability to perform certain tasks or may aggravate pre-existing respiratory disease.

Orehek et al. (1976) obtained dose-response curves for changes in bronchial reactivity to a bronchoconstricting agent after a 1-hour exposure to 0.19 mg/m^3 (0.1 ppm) NO_2. Three of 20 asthmatics showed marked increase in reactivity with 10 others showing smaller increases, for an overall statistically significant effect attributed to this low exposure to NO_2. There has been considerable controversy over the interpretation of these findings, and a similar study recently conducted at the U.S. EPA facilities in North Carolina failed to replicate these results (Heuter, personal communication). Moreover, the health significance of increased bronchial reactivity is open to question. Nonetheless, the study does provide evidence that asthmatics may respond to NO_2 at lower levels than the population at large.

There is considerable uncertainty as to the level at which NO_2 exposure causes symptoms even in healthy adults. The Kerr et al. (1979) study mentioned above suggested that some individuals (particularly asthmatics) may experience symptoms which cause them discomfort, restrict their normal activity or limit their performance, at exposures of 0.94 mg/m^3 (0.5 ppm) NO_2. The symptoms reported in this study have been considered by the EPA (August 1982) to represent adverse health effects.

The EPA (ibid) concluded that chronic bronchitics, asthmatics, and individuals with emphysema constitute groups at risk, even though human evidence directly relating to the latter group is scarce. The EPA (ibid) also concluded that children are particularly sensitive to NO_2, based on the gas stove studies which indicated their increased risk of respiratory symptoms and infection. Their increased risk may be either due to the higher activity level of children which results in greater relative dose of NO_2, and/or to their inherently greater biological sensitivity. In addition, persons with cirrhosis of the liver or other liver, hormonal and blood disorders, or who are taking various drugs, may be sensitive to NO_2 due to their increased systematic, haematological and hormonal alterations after exposure to NO_2. The evidence for these conclusions has been summarized in Table 4.2-19. It has also been surmised that the elderly and people with cardiovascular disease may also be presumed to be more sensitive to the population at large (EPA, September 1982).

Interaction with Other Pollutants and Other Considerations

In view of the uncertainty concerning the lowest adverse effect level and the high biological activity of NO_2, the WHO had recommended in 1977 that a safety factor of 3-5 be applied and that the maximum 1-hour exposure to NO_2 be set at 0.19-0.32 mg/m^3 (0.10-0.17 ppm). They cautioned that this level may still be high in view of the fact that some individuals may be particularly sensitive to this substance, and in view of the biological evidence of the interaction of NO_2 with other air pollutants. Some of the studies relating to the pulmonary function effects of exposure to NO_2, combined with other pollutants, are summarized in Table 4.2-20. It is noteworthy that elevated concentrations of NO_2 may increase the retention of inhaled particulate matter, although the levels at which this occurs has not been determined. The EPA (August 1982) felt that the results reported by von Nieding _et al_. (1977) are difficult to interpret due to questions regarding the health significance of increased bronchoreactivity, some methodological uncertainties and the lack of confirmation by other investigations. In general, it seems that the effects of NO_2 in the presence of other pollutants are additive rather than synergistic (ie. or greater than additive).

Table 4.2-19 **Summary of Potentially Sensitive Groups****

Sensitive Group	Supporting Evidence	References for Supporting Evidence	U.S. Population Estimates
Children	Children under age 2 exhibit increased prevalence of respiratory infection when living in homes with gas stoves. Children up to age 11 exhibited increased prevalence of respiratory infections when living in gas stove homes.	Speizer et al, 1980 Melia et al, 1979	age 0-5 17.2 million* age 5-13 36.6 million*
Asthmatics	Asthmatics reacted to lower levels of NO_2 than normal subjects in controlled human exposure studies.	Kerr et al, 1979 Orehek et al, 1976	6.0 million*
Chronic Bronchitics	Chronic bronchitics reacted to low levels of NO_2 in controlled human exposure studies.	Kerr et al, 1979 Von Nieding et al, 1971 Von Nieding et al, 1970	6.5 million*
Emphysematics	Emphysematics have significantly impaired respiratory systems. Because studies have shown that NO_2 impairs respiration by increasing airway resistance, it is reasonable to assume that emphysematics may be sensitive to NO_2.	Von Nieding et al, 1971 Beil and Ulmer, 1976 Orehek et al, 1976	1.3 million*
Persons with Tuberculosis, Pneumonia, Pleurisy, Hay Fever or Other Allergies	Studies have shown that NO_2 increases airway resistance. Persons who have or have had these conditions may be sufficiently impaired to be sensitive to low levels of NO_2.	Von Nieding et al, 1971 Beil and Ulmer, 1976 Orehek et al, 1976	unknown
Persons with Liver, Blood or Hormonal Disorders	NO_2 induces changes in liver drug metabolism, lung hormone metabolism, and blood biochemistry.	Menzel, 1980 Miller et al, 1980 Posin et al., 1979	unknown

*1970 U.S. Bureau of Census and 1970 U.S. National Health Survey

**Reproduced from E.P.A., Aug. 1982. All subgroups listed are not necessarily sensitive to NO_2 exposure.

See source for complete references.

Table 4.2-20 EFFECTS ON PULMONARY FUNCTION IN SUBJECTS EXPOSED TO NO_2 AND OTHER POLLUTANTS*

Concentration (ppm)	Exposure Duration	Study Population	Reported Effects	References
0.05 NO_2 + .11 SO_2 + 0.025 O_3 (mg/m^3: 0.09 NO_2 + 0.29 SO_2 + 0.05 O_3)	2-Hours	11 healthy subjects	Increased sensitivity to bronchoconstrictor as shown by increases in R_{aw}. No effect on A_aDO_2 or R_{aw} without bronchoconstrictor.	von Nieding et al., 1977
0.50 O_3; 0.50 O_3 + 0.29 NO_2; 0.50 O_3 + .29 NO_2 + 30 CO (mg/m^3: 1.0 O_3; 1.0 O_3 + 0.55 NO_2; 1.0 O_3 + 0.55 NO_2 + 34.4 CO)	4-Hours	4 healthy male subjects	Minimal change in pulmonary function caused by O_3 alone. Effects not caused by NO_2 or CO.	Hackney et al., 1975
0.25 O_3; 0.25 O_3 + 0.29 NO_2; 0.25 O_3 + 0.29 NO_2 + 30 CO (mg/m^3: 0.5 O_3; 0.5 O_3 + 0.55 NO_2; 0.5 O_3 + 0.55 NO_2 + 34.4 CO)	2-Hours	7 male subjects, some believed to be unusually reactive to respiratory irritants	Minimal change in pulmonary function caused by O_3 alone. Effects not increased by NO_2 or CO.	Hackney et al., 1975
50 CO + 5 SO_2; 4.3 NO_2 + 50 CO + 5 SO_2 (mg/m^3: 57.3 CO + 13.0 SO_2; 9.0 NO_2 + 57.3 CO + 13.0 SO_2)	-	3 subjects	Increase in dust retention from 50% to 76% after NO_2 was added to air containing SO_2 and CO.	Schlipkōter and Brockhaus, 1963
0.5 O_3; 0.5 O_3 + 0.5 NO_2 UNDER FOLLOWING CONDITIONS: 1) 25°C, 45% rh 2) 30°C, 85% rh 3) 35°C, 40% rh 4) 40°C, 50% rh (mg/m^3: 1.0 O_3; 1.0 O_3 + 0.94 NO_2)	Rest-60 min. Exercise-30 min. Rest-30 min.	8 young adults	Response found only for O_3; no greater than additive effect or interaction between O_3 and NO_2 was observed.	Horvath and Folinsbee, 1979

*Reproduced from E.P.A., Aug. 1982; see source for complete references

Quantitative Assessments

The effects of NO_2 exposure are summarized in Table 4.2-21, along with the estimates of levels at which these effects may be expected to occur. As is the case for other pollutants, considerable uncertainty still exists with respect to dose-response relationships.

The time-frame of the air quality standard, as discussed in Chapter 3, has been a controversial issue. As mentioned above, the decision in the U.S. to establish only an annual standard with no separate short term standard was based not on the failure to recognize the preponderance of evidence for effects attributable to short term peaks, but on their contention that an annual standard in the range of 94 $\mu g/m^3$ (0.05 ppm) would provide adequate protection against short term peak exposures and would be easier to implement. This approach, therefore, need not be followed in other jurisdictions, and, in fact, indeed is not. A recent report prepared for and endorsed by Health and Welfare Canada (Ferris, 1982) recommended a 1-hour air quality objective of 752 $\mu g/m^3$ (0.4 ppm), noting the negative findings of Hackney et al. (1975) and Nieding et al. (1979), as well as the positive findings of Kerr et al. (1975b) and Orehek et al. (1976). A 24-hour limit of 470 $\mu g/m^3$ (0.25 ppm) was recommended based on the negative findings of Hackney et al. (1975b), Linn et al. (1976), as well as Speizer and Ferris (1973a and b) and Burgess et al. (1973). Finally, the annual concentration of 94 $\mu g/m^3$ (0.05 ppm) was felt to carry minimal risk even for sensitive populations, based on the work of Linn et al. (1976), Cohen et al. (1972), and Bouhuys et al. (1978).

TABLE 4.2-21

COMPILATION OF HEALTH EFFECTS REPORTED IN SELECTED STUDIES OF HUMANS EXPOSED TO LOW LEVELS OF NO_2

NO_2 Concentration					
ppm	mg/m^3	Duration of Exposure	Study Design	Effect	Reference
0.02-0.06	0.04-0.11	Hourly arithmetic mean	Epidemiological (non-smokers)	No effect on pulmonary function or prevalence of chronic respiratory disease	Cohen et al., 1972
0.03-0.05	0.06-0.09	Means of 25 hour averages	Epidemiological (community)	No effect on pulmonary function or symptoms of respiratory disease	Bouhuys et al., 1978
0.03-0.07	0.07-0.13	Median of maximal hourly averages	Epidemiological (office workers)	Respiratory symptoms associated with episodes of smog (SO_2,TSP and oxidants also present)	Linn et al., 1976
0.03-0.28	0.06-0.53	1 hour average	Epidemiological (children)	Decrease in selected pulmonary function tests; significant effect in most sensitive subject at 0.08 mg/m^3 (0.04 ppm)	Kagawa et al., 1976
0.045	0.08	Average winter 1971 levels	Epidemiological (policemen)	No effect on the prevalence of respiratory symptoms or chronic respiratory disease	Speizer and Ferris, 1973a,b
0.05;5	0.09,9.4	2 hours	Controlled experiment	Increased sensitivity to bronchoconstrictors in the presence of other pollutants; no effect on gas exchange or airway resistance	von Nieding et al. 1979
0.04-2.01	0.08-1.07	5-25 min.	Controlled experiment	Possible impairment of dark adaptation	Shalamberidze, 1967
0.04-0.08	0.08-0.16	Mean integrated 24 h values	Epidemiological (children)	Possible decrease in pulmonary function and increase in morbidity	Shy et al., 1970a,b, Pearlman et al., 1971
0.1	0.19	1 h	Controlled experiment	Increased sensitivity to bronchoconstrictors in asthmatics	Orehek, 1976
0.1	0.19	2 min	Controlled experiment	Perception of odour	Feldman, 1974
0.5	0.9	2 h	Controlled experiment	Mild symptoms and changes in pulmonary function in healthy adults, bronchitics and asthmatics	Kerr et al., 1979
0.5-1.0	0.9-1.9	24 h integrated averages	Epidemiological (gas stove homes)	Possible increase in respiratory illness and decrease in pulmonary function in young children	Speizer et al., 1980
0.7-2.0	1.3-3.8	10 min (1 day)	Controlled experiment	Possible increased airway resistance in healthy adults	Suzuki and Ishikawa, 1965
1,2	1.9,3.8	2 h/d for 2 days	Controlled experiment	Small changes in pulmonary function tests and possible increase in respiratory symptoms	Hackney, 1978
1.6,2	3.0,3.8	30 breaths	Controlled experiment	Increase in airway resistance in chronic bronchitics	von Nieding et al., 1971
1-5	1.9-9.4	15-60 min	Controlled experiment	Decrease in blood gas parameters for both healthy adults and bronchitics	von Nieding et al., 1973
2.5,5.0	4.7,9.4	2 h, 14 h	Controlled experiment	Increase in airway resistance	Beil and Ulmer, 1973

4.2.4 Ozone

Introduction

The air quality standards for ozone and other oxidants have been the focus of considerable controversy. Many states in the U.S. had great difficulty in trying to attain the ambient air quality standard of 160 $\mu g/m^3$ (0.08 ppm), and, as a result of a law suit launched by the American petroleum industry, the U.S. EPA was forced to review the basis for the levels set. The evidence for the effects of oxidants on asthmatics on which the U.S. standard had been based came from a study by Schoettlin and Landau (1961) which has since been re-interpreted as showing effects at 500 $\mu g/m^3$ (0.25 ppm) instead of at 200 $\mu g/m^3$ (0.1 ppm) which had originally been thought (EPA, Feb. 1979). Moreover, while most of the clinical experiments were done utilizing exposure to ozone, the U.S. (and other) standards were applicable to total oxidants. Accordingly, the U.S. EPA (ibid) has changed to an ozone standard as described in Chapter 3. Furthermore, the role of exercise in setting standards has been the focus of debate. While there is increasing emphasis on promoting exercise in the population at large, there has been some contention regarding the extent to which "the jogging asthmatic" should also be protected from deleterious effects resulting from ozone exposure. Recent studies such as that conducted by McDonnell et al. (1983) have shed further light on this subject.

The issue of "adaptation" has also complicated the formulation of an ozone standard. A difference between dose-response relationships for Los Angeles subjects as compared to Montreal subjects had been demonstrated (Hackney et al., 1977), with the latter failing to show a no-adverse-effect level. This study has been said to indicate that L.A. subjects exhibited adaptation. Linn and Hackney (1981), however, have shown that adaptation may be a temporary phenomenon and that there is a large variability among individuals regarding their "adaptability" to ozone. Since there are substantial numbers in the general population who are not protected by adaptation, and since adaptation seems to be lost quickly in the absence of very frequent exposures, these authors felt that this phenomenon should not be an important consideration in setting air quality standards.

Although there has been some animal evidence suggestive of adverse effects attributable to chronic low level exposure (Heuter, personal communication 1983; EPA, Feb. 1979), it is generally felt that evidence to qualify this effect is lacking (EPA, Feb. 1979; WHO, 1979; Melton, 1982). Folinsbee et al. (1980) did demonstrate that there is some short-term cumulative effect of exposure to ozone above the threshold for producing acute respiratory effects. They hypothesized that this was due to ozone's ability to increase the sensitivity of the irritant receptors in the airways, which, in turn, potentiates the effect of subsequent ozone exposure. The Advisory Panel of the EPA (EPA, Jan. 1978) arrived at the consensus that the risk of effect can be related to the total dose of ozone delivered to the respiratory tract within a day, (but not over longer periods), and that this risk increased with the frequency of exposures, with the concentration of a single dose, and with the intensity of exercise of exposed subjects. The EPA position is currently under review.

The current state of knowledge will be briefly elaborated.

Nature and Mechanisms of Ozone Toxicity - A Look at the Animal Data: Biochemical and Morphological Abnormalities: Ozone causes damage to biological tissue due primarily to its strong oxidant effect. This provides it with the capability of attacking the double bond in unsaturated hydrocarbons, of significance since human cellular structures, especially plasma membranes, contain unsaturated fatty acids. The reactions of ozone with sulfhydryl groups of amino acids are also well known and important because of the possibility that proteins can be destroyed or immunologically altered by ozone. Nucleic acids or nucleoproteins may also be altered by ozone (Prat et al. 1968).

Ozone's ability to damage membranes and to denature protein explains its powerful effect on human health. The dysfunction it causes is most marked in those cells lining the respiratory tract, since these are the cells in contact with ozone. Irritation of the mucous membranes and lung tissue is thus the chief effect of ozone exposure. Ozone-induced effects on organ systems away from the lung have also been reported. Numerous studies (as summarized by WHO, 1979); EPA, April 1978, and NAS, 1977) have illustrated an impressive variety of biochemical alterations associated with ozone exposures over the range of 200 to 2000 $\mu g/m^3$ (0.1 to 1.0 ppm) (See Table 4.2-22). The EPA (January 1978) judged that effects induced by 200 to 400 $\mu g/m^3$ (0.1 to 0.2 ppm) exposures could possibly be prevented or reversed with increased vitamin E levels in the lung or increased antioxidants at other tissue sites, but, nevertheless, represent the organism's response to stress and may pose a health risk for particularly susceptible individuals. They concluded that levels of 1000 $\mu g/m^3$ (0.5 ppm) and greater have definite toxic potential, but pointed out that there is no sharp dividing line between adaptive responses and potential for pathological consequences. A range of morphological effects has been noted in association with experimental ozone exposures of 400 to 2000 $\mu g/m^3$ (0.2 to 1.0 ppm). These effects varied from seemingly innocuous alterations of alveolar cells to emphysematous changes and terminal bronchiole and alveolar damage. Occurrence of these effects after long term exposure to low concentrations raises the level of suspicion that repeated or chronic exposures may have the potential for inducing similar effects in humans (EPA, January 1978).

Increased Susceptibility to Infection: Increased susceptibility of animals to bacterial infection has been described by several investigations (Coffin et al. 1968; Ehrlich, 1980; Gardner et al., 1974; Miller et al. 1978) following exposure to 200 $\mu g/m^3$ (0.1 ppm) ozone. These reports are consistent with the evidence that establishes indices of infection and/or mortality from bacterial infection as sensitive measures of ozone-induced effects (including Coffin and Gardner

Table 4.2 -22 Experimental animal studies *- ozone*

I. Local effects on the respiratory system

1. *Morphological changes*

Ozone concentration		Length of exposure		Effects	Response[a]	Species	Number of animals a	Reference *
µg/m³	(ppm)	number of days	h/day					
1800	(0.88)	180	24	Epithelial injury seen as early as 4 h after the beginning of exposure; after 3 weeks half of animals died and emphysema-like lesions observed.	n.a.[b]	rat	n.a.	Freeman et al. (1974)
1600	(0.8)	7	24	Walls and interalveolar septa of terminal airways thickened and infiltrated by mononuclear cells.	—	rat	8 (8)	Castleman et al. (1973a)
1200	(0.6)	1	7	Swelling of epithelial alveolar lining cells & endothelium cells with occasional breaks in basement membrane.	—	mouse	32 (13)	Bils (1970)
1100	(0.54)	180	24	Progressive changes in the airway epithelium after 6 days.	—	rat	n.a.	Freeman et al. (1974)
1000	(0.5)	1	6	Immediately after the exposure the number of alveolar cells significantly decreased.	—	mouse	16 (12)	Evans et al. (1971)
800	(0.4)	5 per week × 10 months	6	Emphysematous & vascular-type lesions.	—	rabbit	6 (6)	P'an et al. (1972)
520-2000	(0.26 -1.0)	1	4.7–6.6	Dose-related loss of ciliated epithelium.	—	cat	14 (3)	Boatman et al. (1974)
400	(0.2)	1	2	Degenerative changes in type I cells.	—	rat	n.a.	Stephens et al. (1974)
				2. *Functional changes*				
1400	(0.68)	1	2	No significant increase in flow resistance.	—	guineapig	10 (10)	Murphy et al. (1964a)
1000	(0.5)	1	2	Increase in airways resistance and breathing frequency with decrease in tidal volume.	—	guineapig	10 (10)	Yokoyama (1972a)
520-1000	(0.26 -0.5)	1	4.6	Increased flow resistance.	—	cat	10 (4)	Watanabe et al. (1973b)
400	(0.2)	30	24	Reduction in lung elasticity; increase in lung volume and in alveolar dimensions.	—	rat	44 (44)	Bartlett et al. (1975)
				3. *Biochemical changes*				
1500	(0.75)	1	3	Reduction in activity of benzopyrene hydroxylase (1.14.14.2).	—	hamster & rabbit	25 (95) 8 (15)	Palmer et al. (1971, 1972)
1400-1600	(0.7 -0.8)	7	24	Increased acid phosphatase activity.	—	rat	14 (12)	Castleman et al. (1973b)
1400	(0.7)	5	24	Indication of lipid peroxidation; increase in lysosomal hydrolase activity.	—	rat	33 (20)	Chow & Tappel (1972); Dillard et al. (1972)
1000	(0.5)	1	6	Increased albumen recovery from alveolar spaces.	—	rat	10 (18)	Alpert et al. (1971a)
800-1400	(0.4 -0.7)	1	4	Evidence of formation of lipid peroxides in the lung.	n.a.[b]	mouse	n.a.	Goldstein et al (1969)
500	(0.25)	1	3	Reduced activity of several lysosomal hydrolases.	—	rabbit	6 (6)	Hurst et al. (1970)
400	(0.2)	7	24	Increase in pulmonary mitochondrial oxygen consumption.	—	rat	5–8 (5–8)	Mustafa et al. (1973)
				4. *Effects on the host defence system*				
1200-1600	(0.62 -0.80)	1	17	Inhibition of pulmonary bactericidal activity.	—	mouse	20 (29)	Goldstein et al. (1971b)
1000	(0.5)	210	16	No effect on physical clearance of inhaled particles or on the number of macrophages.	—	rabbit	8 (8)	Friberg et al. (1972)
1000	(0.5)	60	16	Decrease in the clearance of viable *Escherichia coli*.	—	guineapig	18 (18)	Friberg et al. (1972)
800	(0.4)	1	4	Inhibition of bactericidal activity; no additive role of the induced silicosis.	—	mouse	38 (37)	Goldstein et al. (1972)
600	(0.3)	1	3	Impairment of phagocytic properties of pulmonary alveolar macrophages.	—	rabbit	n.a.[b]	Coffin et al. (1968b)
500	(0.25)	1	3	Diminished enzyme activities of alveolar macrophages.	—	rabbit	6 (6)	Hurst et al. (1970)
160	(0.08)	1	3	Increased susceptibility to *Streptococcus*.	15/40 (6/40)	mouse	40 (40)	Coffin et al. (1968a)

[a] Number of animals showing effects/total number of animals; numbers in brackets refer to control groups.

[b] Not available.

Table 4.2-22 (Cont'd)

Experimental animal studies—continued

II. Systemic reactions and other effects

Ozone concentration		Length of exposure		Effects	Response[a]	Species	Number of animals	Reference
µg/m³	(ppm)	number of days	h/day					
				1. *Haematological effects*				
1700	(0.85)	1	4	Formation of Heinz bodies in red cells.	n.a.[b]	mouse	n.a.	Menzel et al. (1975)
1600	(0.8)	8	24	Increase of lysozyme activity in plasma and soluble fraction of lung.	—	rat	8 (8)	Chow et al. (1974)
800	(0.4)	5 per week × 10 months	6	Increase in serum trypsin protein esterase.	—	rabbit	6 (6)	P'an & Jegier (1972); Jegier & P'an (1973)
600	(0.3)	1	1	Inhibition of acetylcholine esterase (3.1.1.7) activity.	—	ox (*in vitro*)	—	P'an & Jegier (1970)
400	(0.2)	1	1–2	Increased sphering of red blood cells.	—	mouse, rabbit, rat (*in vitro*)	—	Brinkman et al. (1964)
400	(0.2)	1	2	Doubling in number of binucleated lymphocytes.	—	mouse	n.a.	Veninga (1970, unpublished)
110	(0.06)	93	24	Decrease in blood choline esterase activity.	—	rat	15 (15)	Eglite (1968)
				2. *Effects on reproduction*				
400	(0.2)	5 per week × gestation period + 1st 3 weeks of life	7	Increase in neonatal mortality.	n.a.	mouse	n.a.	Veninga (1967)
200–400	(0.1–0.2)	5 per week × 3 weeks	7	Increase in neonatal mortality.	n.a.	mouse	n.a.	Brinkman et al. (1964)
				3. *Behavioural changes*				
1000–2000	(0.5–1.0)	1	1	Decrease in the amplitude of evoked response to flash.	—	rat	3 (3)	Xintaras et al. (1966)
1000	(0.5)	1	0.5	Increase in simple and choice reaction time.	—	rhesus monkey	4 (4)	Reynolds & Chaffee (1970)
1000	(0.5)	1	0.75	Significantly reduced motor activity.	—	rat	12 (12)	Konigsberg & Bachman (1970)
600–1000	(0.3–0.5)	60	intermittently (variable intervals)	Increase in time to learn specific tasks.	—	rat	6 (6)	Litt et al. (1968)
400	(0.2)	1	6	Reduction in spontaneous running activity.	—	mouse	9 (9)	Murphy et al. (1964a)
				4. *Miscellaneous extrapulmonary changes*				
1500	(0.75)	1–2	4–8, 24	Histological changes in parathyroid gland.	—	rabbit	16 (16)	Atwal & Wilson (1974); Atwal et al. (1975)
400	(0.2)	21	5	Structural changes in myocardial muscle fibres.	—	rabbit & mouse	n.a.	Brinkman et al. (1964)
400	(0.2)	1	5	Increase in chromosomal breaks in circulating lymphocytes.	—	hamster	8 (4)	Zelac et al. (1971a, b)

[a] Number of animals showing effects/total number of animals; numbers in brackets refer to control groups.

[b] Not available.

*Reproduced from W.H.O., 1979. See source for complete reference

1972 and Goldstein et al. 1971a and b, 1972 and 1974). There is also considerable evidence that additional stress such as heat, exercise or a combination with other pollutants may enhance the effect of ozone on susceptibility to infection, and may thereby lower the ozone dose at which the subject will be adversely affected. The EPA (1978) noted the capacity of ozone to induce irritation of the major bronchi in man at ozone concentrations in the range of 500 $\mu g/m^3$ (0.25 ppm).

They also alluded to the fact that biochemical and cellular alterations described in rodents as ozone-induced effects have also been demonstrated in humans when viral infection precedes the onset of bacterial pneumonia. The EPA (1978) therefore concluded that it is reasonable to expect that ozone can predispose human beings to infection, although the exposure levels associated with such effects may be different.

Genetic and Teratogenic Potential; Zelac (1971 a&b) reported: chromosomal abnormalities in peripheral leukocytes in hamsters after 5 hours of exposure to 400 $\mu g/m^3$ (0.2 ppm). Gooch et al. (1976) however, failed to confirm these findings. Merz et al. (1975) did report similar abnormalities in humans exposed to 1000 $\mu g/m^3$ (0.5 ppm) ozone, but again there has been no replication of these findings. Moreover, the health significance of chromosomal aberrations is open to debate. The EPA (ibid) cited the observation of Veninga (1967) of increased neonatal deaths and congenital abnormalities in newborn mice, as grounds for raising the index of concern over the potential teratogenic effects of ozone.

Health Effects of Ozone Exposure: Clinical and Epidemiological Data

Tables 4.2-23 and 4.2-24 summarize the human studies examining ozone or oxidant exposure. It can be seen that ozone or other oxidants impair pulmonary function, cause respiratory and other

TABLE 4.2-23

RESULTS REPORTED IN SELECTED CONTROLLED HUMAN STUDIES OF OZONE EXPOSURE

A. Ozone

Ozone Concentration		Duration of Exposure	Effects	Reference
ppm	µg/m³			
0.008-0.02	15-40	Immediate	Threshold of odour perception	Eglite, 1968
≥0.1	≥200	Working hours for 123 d	Increased eye irritation	Richardson and Middleton, 1957, 1958
0.1	200	2 h	Increased airway resistance in healthy subjects under light intermittent exercise	von Nieding et al., 1977
0.1	200	2 h	Decreased O_2 in arterialized blood and increased airway resistance (using nonstandard measurement techniques)	von Nieding, et al., 1979
0.12 -0.40	240-800	2.5 h	Increased pulmonary function under intermittent vigorous exercise, symptoms at all exposures	McDonnell et al., 1983
0.15 -0.30	300-600	1 h	Symptoms of discomfort reported and discernible (but not statistically significant) changes in respiratory patterns under vigorous exercise	Delucia and Adams, 1977
0.2 -0.25	400-500	2 h	No statistically significant changes in pulmonary function, but slight increase in symptoms and biochemical changes in asthmatic patients	Linn et al., 1978
0.25	500	2 h	Small changes in lung function under light intermittent exercise	Hazucha et al., 1973
0.25	500	2 h	No important lung changes in "reactive" subjects under light intermittent exercise	Hackney et al., 1975
0.2 -0.4	400-800	2 h	Increased bronchial reactivity	Nadel et al., 1979
			Decreased pulmonary function	Folinsbee et al., 1978
0.30	600	1 h	Symptoms of discomfort and statistically significant changes in pulmonary function under vigorous exercise	Delucia and Adams, 1977
0.37	740	2 h	Symptoms of discomfort and significant changes in lung function under light intermittent exercise	Hazucha et al., 1973 Folinsbee et al., 1975
0.37	740	2 h	Significant increase in total airway resistance under light intermittent exercise	Hackney et al., 1975
0.5	1000	6 h	Significant change in airway resistance and increased symptoms (dry cough and chest discomfort)	Kerr et al., 1975

B. Ozone and Other Pollutants

ppm	µg/m³	Duration of Exposure	Effects	Reference
0.025 0.05 0.1	50 NO_2 100 SO_2 260	2 h	Increased sensitivity bronchoconstriction	von Nieding et al., 1977
0.37 0.37	730 SO_2 1000	2 h	Lung function changes greater than additive	Hazucha and Bates, 1975

TABLE 4.2-24

COMPILATION OF RESULTS FROM SELECTED STUDIES OF THE EFFECTS OF COMMUNITY EXPOSURE TO OZONE

Ozone Concentration ppm	Ozone Concentration $\mu g/m^3$	Concentration Measurement	Effects	Reference
0.06	125	Mean daily maximum	Increase in hospital admissions for respiratory disease	Bates and Sizto, 1983
0.01-0.03	20-60	Mean hourly maximum	Lung function parameters were significantly correlated with hourly O_3 concentrations in the 2 hours prior to testing. Other pollutants also correlated. No threshold determined	Kagawa *et al.*, 1976
≥0.03	≥60	Mean hourly maximum	High correlation with increased risk of asthma attack. Other pollutants also correlated. No threshold determined	Whittemore and Korn, 1980
0.08	160	Mean hourly maximum	Significant correlation with cough and eye irritation in sensitive people	Zagraniski *et al.*, 1979
0.03-0.30	60-590	Hourly average	Significant correlation with decreased athletic performance, at least for values above 0.2-0.3 mg/m^3	Wayne *et al.*, 1967
≥0.15	≥300	Daily maximum	Increased frequency of respiratory symptoms and headache	Makino and Mizoguchi, 1975
≥0.25	≥500	Daily maximum	Increased frequency of asthma attacks	Schoettlin and Landau, 1961
≈0.50	≈1000	Hourly maximum	Increased symptoms began at 100 $\mu g/m^3$ (headaches); 300 $\mu g/m^3$ (eye irritation); 530 $\mu g/m^3$ (cough); 580 $\mu g/m^3$ (chest discomfort). Threshold determination questionable.	Hammer *et al.*, 1974

symptoms, decrease performance, increase susceptibility to infection, and cause exacerbation of asthma or aggravation of other chronic respiratory conditions. Each of these effects will be addressed below, with the issue of susceptible populations discussed in a later section.

Impairment of Lung Function: Based on the findings of Hazucha and Bates (1975), Hazucha et al. (1973) and Folinsbee et al. (1977), the EPA (1978) concluded that there is very convincing evidence that 740 $\mu g/m^3$ (0.37 ppm) ozone has a deleterious effect on lung function of lightly exercising subjects. The EPA (ibid) felt that the study of Delucia and Adams (1977) raises the possibility that an ozone concentration of 600 $\mu g/m^3$ (0.30 ppm) exerts an effect on lung function of healthy subjects exercising strenuously and that some healthy individuals may even be affected at concentrations as low as 300 $\mu g/m^3$ (0.15 ppm). However, the EPA felt that the finding of deleterious effects on lung function at levels of 200 $\mu g/m^3$ (0.1 ppm) reported by Nadel and Goldsmith (1969) and von Nieding et al. (1977) were unreliable for methodological reasons. The studies of Kagawa and Toyama (1975), Delucia and Adams (1977) and Hackney et al. (1975) illustrate the increased significance of ozone-induced lung function impairment of patients with respiratory disease. These and other studies relating to the oxidant effects in asthmatics (such as Schoettlin and Landau, 1961; Whittemore and Korn, 1980; Bates and Sizto, 1983; Zagraniski et al. 1979) will be discussed below. Of considerable importance is the recent clinical study by McDonnell et al. (1983) in which small, but statistically significant changes in pulmonary function (FVC - forced vital capacity, FEV - forced expiratory volume, and $FEF_{25\%-75\%}$ - forced expiratory flow between 25% and 75% of the forced expiratory vital capacity), were observed in exercising healthy young males (ie. non-asthmatics) at levels as low as 240 $\mu g/m^3$ (0.12 ppm). The study also showed a large difference in individual response, indicating that even "non-asthmatics" have a variable degree of airway reactivity to ozone. The results of this study will, no doubt, have far-reaching consequences in regulatory decisions.

Clinical Symptoms of Ozone Exposure: It has been noted (EPA, 1978) that in nearly all experimental studies in which ozone exposure has been sufficient to produce lung function changes, most subjects reported respiratory symptoms. Hammer et al. (1974), attempting to obtain threshold estimates for the various symptoms, reported chest discomfort at 500-580 $\mu g/m^3$ (0.25-0.29 ppm), cough at 600-780 $\mu g/m^3$ (0.30-0.39 ppm) and headache at 300-380 $\mu g/m^3$ (0.15-0.19 ppm). Several Japanese studies (Kagawa and Toyama, 1975, 1976) indicated increased rates of sore throat, dyspnea and headache on days when oxidant concentrations exceeded 200 $\mu g/m^3$ (0.10 ppm). Early studies had reported steadily increasing rates of eye irritation at 200 to 900 $\mu g/m^3$ (0.10 to 0.45 ppm) oxidant concentration, although Hammer et al. (1974) reported this effect at 300-380 $\mu g/m^3$ (0.15-0.19 ppm). Another recent study (Zagraniski et al., 1979) demonstrated significantly increased occurrence of cough and eye irritation at even lower levels. The EPA (Jan.1978) felt that the attempts (e.g. by Hammer et al., 1974) to determine thresholds for ozone-induced symptoms were inappropriate and violated biological evidence for nonlinear dose-response relationships. The recent experimental findings of McDonnell et al. (1983) that even healthy young males have a definite increase in cough at 240 $\mu g/m^3$ (0.12 ppm) corroborate the wisdom of this approach.

Decreased Resistance to Infection: The animal evidence that ozone exposure predisposes to infection is reinforced by the Durham study (1974) in which increased illness was reported in college students following periods of elevated pollution (with peak oxidant being the pollution variable most strongly associated with illness).

Asthma Attacks: Despite some confusion in the early interpretations of the Schoettlin and Landau (1961) asthma study, it seems that significantly more asthma episodes occurred on days when peak oxidant concentrations exceeded 500 $\mu g/m^3$ (0.25 ppm), associated with average maximum hourly oxidant concentrations of about 400 $\mu g/m^3$ (0.20 ppm). The EPA (June 1978) felt that the evidence supported the statement that a proportion of asthmatics will be affected by maximum hourly oxidant concentrations of 400 μg/m^3 (0.20 ppm), and that the effect is likely to occur at concentrations in the range of 300-500 μg/m^3 (0.15-0.25 ppm) in some asthmatics or other persons with sensitive airways. The results obtained in a recent study by Whittemore and Korn (1980), as illustrated by Figure 4.2-10, also show that the risk of having an asthma attack indeed increases as the levels of oxidant increases (with all other variables held constant). Even more recently, Bates and Sizto (1983) released their findings on the relationship between air pollutant levels and hospital admissions in southern Ontario. They found high statistically significant associations between excessive respiratory disease admissions for the summer months and SO_2, temperature and ozone. The average ozone level during this period was only 62.8 ppb (126 μg/m^3).

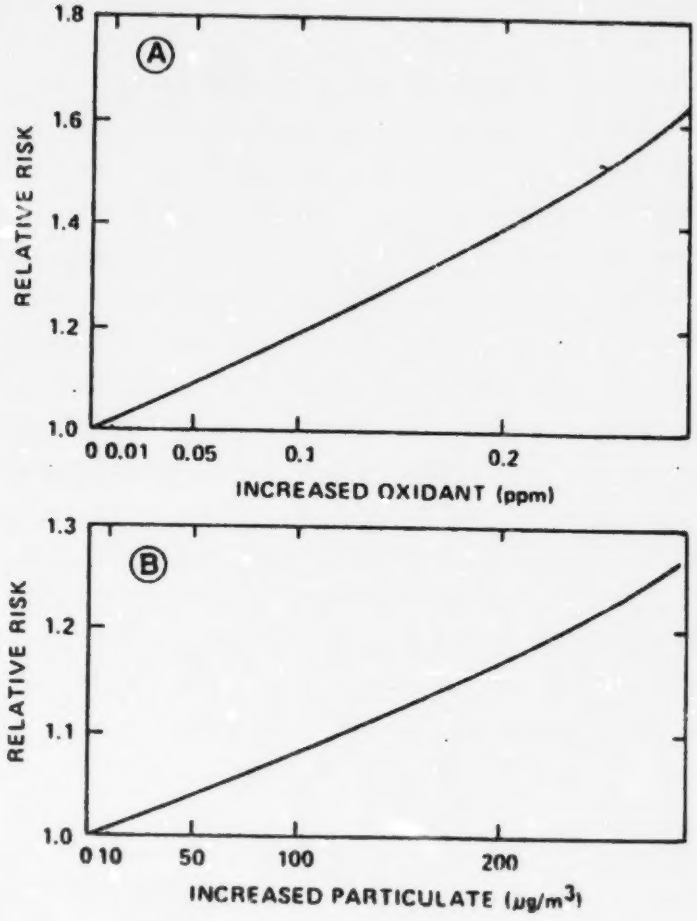
Impaired Athletic Performance: Wayne et al. (1967) demonstrated a high correlation between hourly oxidant concentration and the proportion of runners that fail to improve running time. The oxidant concentrations ranged from 60-600 μg/m^3 (0.03-0.30 ppm), although as the EPA has pointed out (EPA, June 1978), the data do not suggest a plateau in the dose-response function. They further noted that Folinsbee et al. (1977) documented a decline in maximal oxygen uptake in healthy young subjects exercising under controlled experimental exposure to 1500 μg/m^3 (0.75 ppm) ozone, thus suggesting a mechanism for the effect on athletic performance.

Particularly Susceptible Populations

Clinical and epidemiological studies have shown that people with chronic obstructive airways disease, particularly asthmatics, appear most sensitive to changes in ozone concentration. This sensitivity results from the fact that their airways are hyper-reactive to irritants such as ozone. Recent studies (McDonnell et al., 1983) have shown that even non-asthmatics may have hyper-reactivity to ozone.

Concern has been expressed (EPA, Jan. 1978) that ozone exposure in young children may compromise lung development in view of the findings of Bartlett et al. (1974) which demonstrated a reduction in lung elasticity and overdistension of lungs of young rats exposed to 400 μg/m^3 (0.2 ppm) ozone for 30 days. The epidemiological evidence for the effect

Figure 4.2-10 Relative Risk (i.e., odds ratio) of Asthma Attack Corresponding to Specified Increases in Oxidant Level (a) and Particulates (b) With All Other Variables Held Fixed (the odds ratios were computed using the summary fixed effects coefficients 1.66 for oxidant and 0.79 for particulates)*



*Reproduced from Whittemore and Korn, 1980 of oxidants in the range of 200-500 μg/m^3 (0.1-0.25 ppm) to the lung function in children was also cited by the WHO (1979) as cause for concern.

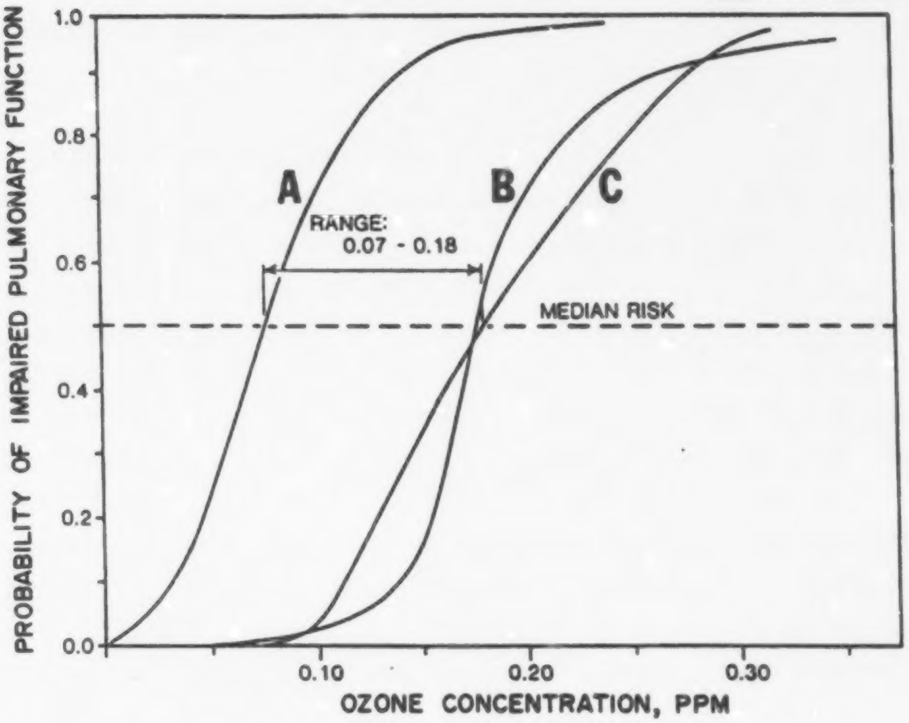
Studies have indeed established that exercise increases the ozone dose that reaches the target tissues in the respiratory tract. Therefore, people engaging in physical activity are particularly vulnerable to the effects of ozone.

Quantitative Assessments and Other Issues Relevant for Standard-Setting

Bates and Hazucha (1973) have shown that sulfur dioxide, at concentrations of 962 μg/m^3 (0.37 ppm), enhances the effects of ozone. There have been differences of opinion as to whether this should argue in favour of providing a margin of safety (EPA criteria document; U.S. EPA, June 1978, for example). Moreover, for ozone even moreso than for substances in general, the no-adverse-effect level depends on the particular subjects who have been studied and the circumstances under investigation. Adverse health effect thresholds for sensitive persons are difficult to detect experimentally and studies performed on small numbers of healthy subjects are not likely to predict the response of sensitive groups. Further, it is noted that the margin of difference between ozone concentrations that produce serious toxicological effects in animals as well as symptomatic and lung function changes in humans, and ambient levels of ozone, is much smaller than for any other atmospheric pollutant. As noted by the EPA Advisory Panel (EPA, June 1978), exposures above maximum background levels of 100-120 μg/m^3 (0.05-0.06 ppm) may well be associated with some increased health risk.

With these notes of caution, Table 4.2-25 summarizes the probable effect levels estimated by the EPA's health panel as well as those derived from estimates determined from interviews with health experts (EPA, Feb. 1979). Wright (1982) illustrates that subjective

Figure 4.2-11 REDUCTION IN PULMONARY FUNCTION RELATED TO OZONE EXPOSURE (FOR MOST SENSITIVE 1% OF THE POPULATION*



*Reproduced from Wright, 1982

Table 4.2-25 PROBABLE EFFECT LEVEL ESTIMATES—*Estimates for sensitive population segments* *

[Parts per million], Ozone

	Aggravation of asthma, emphysema, and chronic bronchitis	Reduced resistance in bacteria infection (animal studies)	Reduction in pulmonary function	Chest discomfort and irritation of the respiratory tract
Health panel judgment of effect level.	0.15-0.25	(¹)	0.15-0.25	0.15-0.25
Probable (median) effect level as estimated from interviews with health experts	0.17 (0.14-0.25)	0.18 (0.07-0.38)	0.15 (0.07-0.18)	0.15 (0.11-0.18)

¹Not available.

* REPRODUCED FROM
EPA, 1979

encoding technique for "category 3" effects (i.e. impaired pulmonary function) as shown in Figure 4.2-11. He points out that while the curves of Experts B and C indicate a 90% probability of adverse effects only above 520 μg/m^3 (0.26 ppm), Expert A's curves does so below 280 μg/m^3 (0.14 ppm). Among his many criticisms of this process, he felt that this discrepancy was primarily due to the failure to define the precise activity level of the hypothetical subject being exposed.

The recent results of Bates and Sizto (1983) are, however, particularly disconcerting in view of the low levels of exposure found to be capable of increasing the rate of admissions for respiratory disease. The fact that on the basis of the Los Angeles data, this strong association would not have been expected, led the authors to postulate three factors which might influence morbidity in Ontario by comparison with Los Angeles. First, the adaptation issue was again identified, indicating that the Ontario population is more sensitive to ozone than the Los Angeles population. Secondly, they felt that the lower ambient temperatures in Ontario, compared to Los Angeles, may mean that more individuals are engaging in out-of-doors exercise, which, as indicated, may potentiate ozone-induced effects. Thirdly, they felt that the higher levels of particulate pollution may be important.

Bates and Sizto (1983) noted that some of the elevations in respiratory admissions involved increases of about 70% on given days, or 28 additional respiratory admissions compared to a normal average of 40. Firstly, they pointed out that this equated to about 0.01% of the 5% most "reactive" individuals and that it may safely be assumed that for each of these hospital admissions, there are several patients who attended a hospital outpatient department and several more who consulted their own physicians - resulting in a considerable economic burden of pollution-related disease. While dose-response curves for the various health effects of ozone are subject to methodological weaknesses, findings such as those of Whittemore and Korn (1980) and Bates and Sizto (1983), are worthy of considerable attention.

4.2.5 Diesel Exhaust Particulates

Introduction

As mentioned in previous chapters, as increasing attention has been directed to fuel-efficiency over the last few years, greater use is being made of diesel engines. The EPA has estimated that by the end of the century, as much as 25% of the cars on the road may be diesel (EST, 1980). In addition to emitting gas phase compounds that have both direct and secondary effects in the atmosphere (as discussed in the other sections of this chapter), diesel engines emit a considerable amount of particulates of both solid (soot) and liquid (aerosol) type.

The high particulate emissions of the diesel have been the focus of concern regarding the health effects of this engine. Studies in 1977 proved that diesel passenger cars produce particulate, 50 to 80 times their gasoline-fueled counterparts (Springer 1981). The particles, as mentioned, are of respirable size (0.1 to 2 μm diameter; see Chapter 2), capable of penetrating deep into the lungs. Thus, one of the health concerns regarding diesel emissions relates to its possible contribution to morbidity and mortality from chronic lung disease (EHL 1980). The soot particle also provides a surface for the aerosol to condense or adsorb. Some well known carcinogens, such as benzo(a)pyrene are among the materials present in diesel particulates. Consequently, the major cause for concern regarding these emissions relates to their possible role in causing cancer.

Uncertainty regarding actual risk rests on the fact that the hydrocarbon substance attached to the particle can change from moment to moment, depending upon the engine design, horsepower, type of fuel, operating load, speed, engine maintenance, engine lubrication and other factors (Weisenberger 1981). At one time, the objection was raised that the hydrocarbons may have become attached to the particulates as they were filtered out to study them; however, it is now acknowledged (ibid) that they are truly attached as a result of the combustion process. Present controversy revolves around the fact that the mutagenic activity of the extract depends in part upon the choice of solvent (ibid), and the question of the relevance of these bacterial findings to humans.

Clearly, quantification of risk attributable specifically to diesel particulates is not possible at present. Nonetheless, it is useful to briefly review the evidence regarding the deleterious effect of diesel particulates on pulmonary function, as well as its role in mutagenesis and carcinogenesis so that the nature of the hazard could be taken into consideration in pressing regulatory decisions that cannot await definitive results.

Effects on Pulmonary Function

Studies of the Effects of Diesel Particulates: An important factor in assessing the potential health effects of diesel emissions relates to the extent to which inhaled particles deposit in the respiratory system and how long they remain before being cleared. Various animal studies have shown that during chronic exposure, the amount of diesel particulate indeed builds up in the lungs over time (Soderholm 1981), with pulmonary deposition relatively high and retention times relatively long (Wolff et al. 1981). The carbonaceous particles deposited in the respiratory airways are phagocytized (engulfed) by the pulmonary alveolar macrophage (immunological cells) and are eventually swept up the respiratory tract via the mucociliary escalator (hair-like cells that line the airways), or cleared through lymphatic channels. Lung clearance via lymphatics results in an accumulation of particles in the regional lymph nodes, and data suggest that the presence of hydrocarbons or carbonaceous diesel particles in high doses might affect immune function (Dziedzic 1981). The pathological description of pulmonary responses to diesel exhaust, as summarized by Moorman et al. (1981), include the observations of (1) marked accumulation of black pigment laden macrophages in the tissue around blood vessels and respiratory bronchioles; (2) hyperplasia of the alveolar lining cells (thickening of the lining of the air sacs) with focal thickening of the surrounding lung tissue; (3)

interstitial pneumonitis (inflammation) and (4) traces of, or no emphysema or peribronchiolitis (Wiester et al. 1980; Karagiones et al., 1981). These pathological findings were felt by Moorman et al. (1981) to be consistent with their physiologic findings of restrictive lung disease in cats exposed for two years to diesel exhaust.

The few studies that have been conducted relating diesel exhaust exposure and pulmonary function in humans (Jorgenson and Svensson 1970; Battigelli 1965; Battigelli et al. 1964) have failed to document an adverse effect. Pattle et al. (1957) concluded that the animal deaths related to diesel emissions were caused by high levels of NO_2, CO and acrolein-type irritants present in the emissions. Gaseous contaminants (CO, NO_x, CO_2, SO_x) and light hydrocarbons, however, were present in a more recent study by Wiester et al. (1980) in concentrations beneath those associated with definable adverse effects. They found that at ambient chamber temperatures, approximately one-half of the hydrocarbons were adsorbed to particulates. In addition, small quantities of NO_x and SO_x were also adsorbed. Wiester et al. (1980) therefore suggested that these "respirable pockets" of highly concentrated irritable substances condensed onto particles may be the source of the irritation effects noted. This logical hypothesis helps explain the difficulty in attempting to evaluate the pulmonary effect of diesel particles per se.

Health Effects of Particulate Matter (TSP): The morbidity and mortality associated with acute exposure to suspended particulates generally, usually in combination with sulfur dioxide, have been summarized by EPA (1980) as shown in Table 4.2-26. From these studies, the minimum air level of total suspended particulates (TSP), as well as SO_2, at which acute mortality increases, was projected by the EPA (April, 1980) to be 300-500 $\mu g/m^3$. The EPA noted that worsening of symptoms in bronchitis patients and increased hospital admissions in Britain was reported to occur at TSP (and SO_2) levels of 300 $\mu g/m^3$ or more, although a U.S. study found exacerbation of symptoms among bronchitics at 200 $\mu g/m^3$ TSP (and 100 $\mu g/m^3$ SO_2) and increased asthma attacks at 150 $\mu g/m^3$ TSP (and 200 $\mu g/m^3$ SO_2). Health effects of chronic exposure to SO_2 and particulate matter are summarized in Table 4.2-27. It should be noted that respirable suspended particulates (RSP), ie.

Table 4.2-26 SUMMARY TABLE - ACUTE EXPOSURE EFFECTS OF TOTAL SUSPENDED PARTICULATES (TSP) AND SULPHUR DIOXIDE (SO_2)***

			24-hour average pollutant levels at which effects appear	
Type of Study	Reference	Effects observed	TSP (μg/m³)	SO_2 (μg/m³)
Mortality (episodic)				
British	Table 14-1	Excess deaths	546*	994
Dutch	Table 14-2	Excess deaths	300-500	500
Japanese	Table 14-2	Excess deaths	285	1800
USA	Table 14-2	Excess deaths	570 (5 CoH)	400-532 (1 hr max: 2288)
(Non-episodic)	Martin and Bradley	Increases in daily mortality	500*	300
	Martin	Increases in daily mortality above the 15 moving average	500*	400
	Glasser and Greenburg	Increases in daily mortality	350-450**	524
Morbidity	Martin	Increases in hospital admissions for cardiac or respiratory illness	500*	400
	Lawther et al.	Worsening of health status among 195 bronchitics	344* (250 BS)	300-500
	Greenberg et al.	Increased cardio-respiratory ER visits	357** (260 BS)	715
	Lawther et al.	Increased clinical condition in CB patients	529* (400 BS) 344* (250-350 BS)	450 300
	Stebbings and Hayes	Increased symptoms in chronic bronchitis (CB) patients	200 (60 RSP) (12SS) 8 SN)	100
	Cohen et al.	Increased AS attacks	150 (20SS)	200
	McCarroll et al.	Increased ARI daily inc/prev	160* (1.2 COH)	372
	Cassell et al.	Increased ARI average daily inc/prev	205* (2 COH)	452
	Stebbings and Fogleman et al.	Decreased $FEV_{0.75}$ (children)	700	300

* Converted from BS (British Smoke).

** Converted from CoH (Coefficient of Haze)

RSP:	Respirable Suspended Particulates
SN:	Suspended Nitrates
SS:	Suspended Sulphates
ER:	Emergency Room
CB:	Chronic Bronchitis
AS:	Asthma
ARI:	Acute respiratory infections
inc/prev:	incidence/prevalence

*** Reproduced from E.P.A., April 1980. See source for complete references.

Table 4.2-27 . SUMMARY TABLE - CHRONIC EXPOSURE EFFECTS OF TOTAL SUSPENDED PARTICULATES (TSP) AND SULPHUR DIOXIDE (SO_2)**

Type of Study	Reference **	Effects observed	Annual average pollutant levels at which effect occurred	
			TSP ($\mu g/m^3$)	SO_2 ($\mu g/m^3$)
Mortality (geog.)	Winkelstein	Increased mortality	125-140	not significant
	Zeidberg and colleagues	Increased mortality	55-60	30
Morbidity				
Longitudinal and cross-sectional	Ferris et al.	Higher rate of respiratory symptoms; and decreased lung function	180	55
Cross-sectional (2 areas)	Sawicki (1972)	More chronic bronchitis, asthmatic disease in smokers; reduced FEV%	250*	125
Cross-sectional study of school-children in 4 areas	Lunn et al.	Increased frequency of respiratory symptoms; decreased lung function in 5-year olds	260*	190
Follow-up of school-children in 4 areas	Douglas and Waller	Increased lower respiratory tract infection	197* (130 BS)	130
Cross-sectional study of children in 4 areas	Hammer et al.	Increased incidence of lower respiratory diseases	85-110	175-250
Cross-sectional study of high school children in 2 areas	Mostardi and colleagues	Lower FVC, FEV$_{0.75}$ and maximal oxygen consumption	77-109	96-100
Cross-sectional (multiple areas)	Lambert and Reid	Increased respiratory symptoms	160* (100 BS)	100-150
Cross-sectional (3 areas)	Goldberg et al.	Increased CRD	78-82	69-160
Cross-sectional (4 areas)	House et al.	Increased CRD	70 (15SS)	100-150
Cross-sectional and Long (2 areas)	Sawicki and Lawrence (1977)	Increased Prev CB and AS Increased persistance, Males 31-50; Increased incidence, Females, some ages	169*	114-130
Cross-sectional (3 areas)	Rudnick	Increased respiratory symptoms in boys. Increased Rh in girls	221-316* (150-227 BS)	108-148
Cross-sectional and retro-long in 4 areas (children)	Nelson et al.	Increased LRD	70	107
Cross-sectional 2 areas	Hammer	Increased LRD	133 (SS=14)	<25
Cross-sectional 3 areas (children)	Shy et al.	Decreased adjusted FEV$_{.75}$ in children > 8 years	78-82	69-160
Cross-sectional 2 areas (children)	Shy et al. Chapman et al.	Decreased adjusted FEV$_{.75}$	96-114 (45 RSP)	(= and low)

* Converted from BS (British Smoke)

**Reproduced from E.P.A., 1980; see source for complete references

geog. - geographical
CRD - Chronic respiratory disease
LRD Lower respiratory disease
RSP - Respirable suspended particulates
BS - British smoke
SS - Suspended sulphates

particles of less than 10 μm have been measured in only a few American epidemiology studies, eg. those by Hammer (1977), Stebbings and Hayes (1976), Shy et al. (1973) and Chapman et al. (1973). As noted by the EPA (1980), the latter study demonstrated pulmonary impairment in children in an area with high pollution with RSP of 45 $\mu g/m^3$ (96 to 114 $\mu g/m^3$ TSP) and very low SO_2, suggesting that RSP of 45 $\mu g/m^3$ may be important.

Numerous methodological difficulties (especially the need to control for a multitude of possible confounding or covarying factors) have hindered the elucidation of dose-response relationships from studies such as these. Figure 4.2-12 depicts the divergence of opinion between various reviews (Holland et al. 1979; WHO 1979; EPA 1980 and various others) in their evaluation of the literature. Suffice it to conclude that there is strong evidence that exposure to particulates has an adverse effect on health and that diesel emissions containing a large amount of respirable particles would certainly be expected to contribute to the adverse effects noted.

Cancer Risk

The major health concern about diesel emissions per se is that its constituents may be cancer-causing. The polycyclic aromatic hydrocarbons (PAH) found in diesel emissions were listed in Chapter 2. The carcinogenic potential of many of these PAH's has been amply demonstrated in many species of laboratory animals following oral, skin and intratracheal administration (IARC 1973). In recent years, Huisingh et al. (1978) demonstrated that diesel particulates had mutagenic activity in the Ames microbial assay (a standardized short term test which estimates the mutagenic and carcinogenic potential of some chemicals). The mechanisms by which diesel particulates induce mutations have since been further elucidated (King et al. 1981; Lewtas 1981; Pereira 1981, and others). The carcinogenic importance of the class of chemicals called nitroarenes found in diesel effluent has recently been reviewed by Rosenkrantz (1982). Although there is still considerable uncertainty, it has been observed (EST 1980) that (1) lung cancer appears to be more prevalent in diesel-exposed hamsters than in controls, (2) cultured mouse cells, salmonella and yeast cells

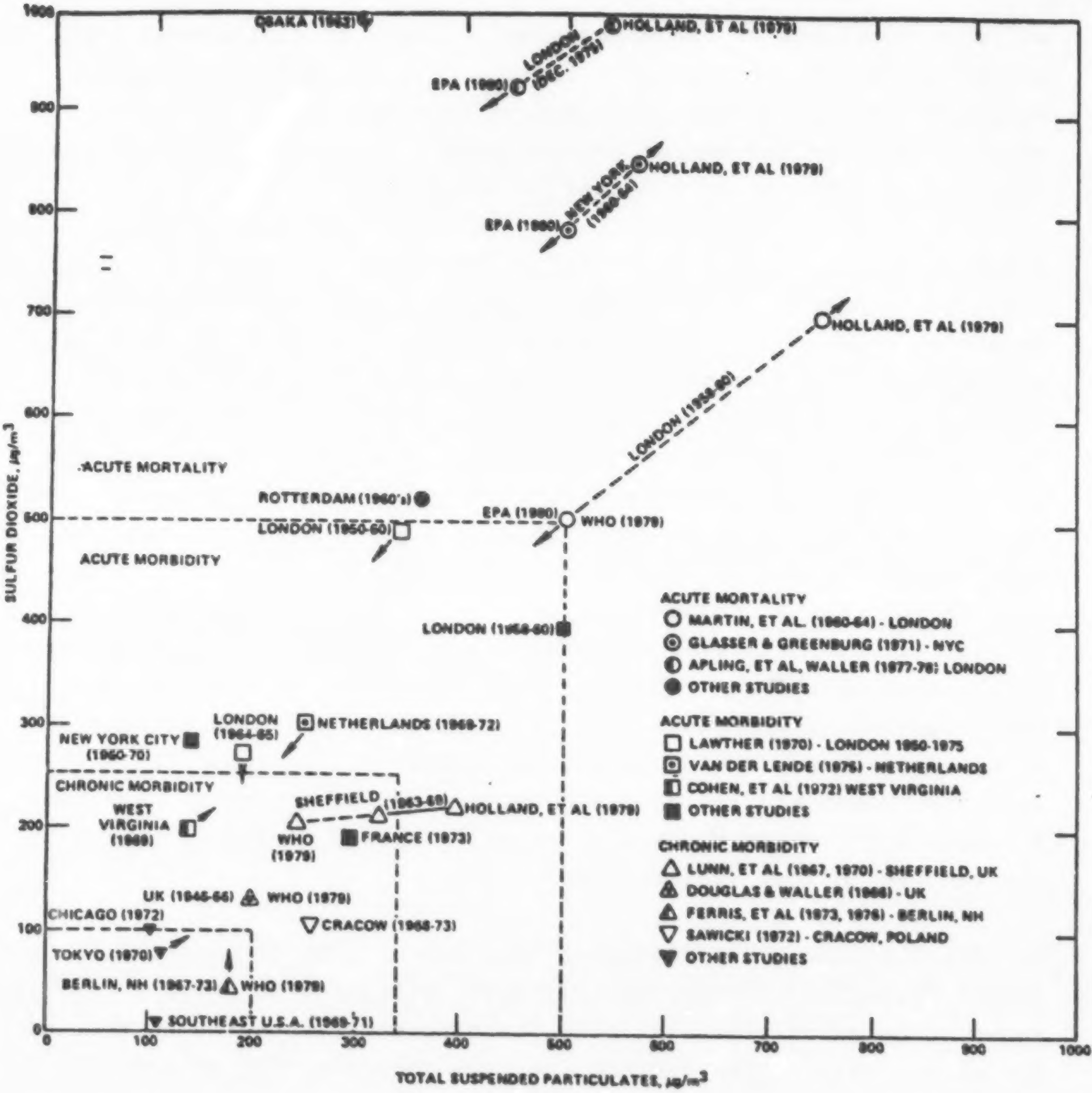


Figure 4.2-12 Comparison of interpretations of studies* evaluated by Holland et al. (1979),[301] WHO (1979),[312] or other reviews such as those in the NRC/NAS documents[307,308] and the present chapter. Aside from the British studies noted for London and Sheffield[301] and the 1960-64 New Youk City mortality study, Holland et al.[301] either ignored the other studies shown or evaluated them as being invalid based on methodological flaws or reinterpretation of their findings. "OTHER STUDIES" not specifically identified in the above key include those reported by: Gervois et al.[61] □ France (1973); Martin[16] □ London (1958-60); Mostardi et al.[117,258] ▽ Chicago (1972); Hammer[113,257] ▽ Southeast USA (1969-71); Suzuki and Hitosugi ▽ Tokyo (1970). The dashed lines depict WHO (1979)[312] conclusions regarding SO_2 and particulate levels associated with acute (24-hr) mortality, acute morbidity, and chronic (annual) morbidity.

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*Reproduced from E.P.A., 1980.

all show mutations or DNA damage when exposed to dichloromethane extracts, and (3) perhaps most far-reaching, diesel particulates were found to be 10 times more active in mutagenicity tests than extracts of gasoline engine particulates. The significance of these findings is, of course, a matter of contention.

Epidemiological studies have also demonstrated increases in respiratory tract or skin cancers in workers exposed to many of the PAHs found in diesel emissions. The carcinogenic potential of soot, coal gas and tar, coke combustion products and mineral oils have been thoroughly reviewed (e.g. NAS 1972; IARC 1973; Cole and Goldman 1975). The epidemiological data on carcinogenic effects of diesel exhaust per se are, however, quite limited and inconsistent. Schenker (1980) reviewed the evidence, as summarized in Table 4.2-28. He pointed out that because of the difficulty in distinguishing gasoline from diesel exhaust exposure in community studies, the data obtained in occupational studies are of particular importance. In noting the inconsistency of the earlier studies of bus and train workers, he stressed that these were all undertaken before diesels had been in use long enough for an adequate duration of occupational exposure. He commented that the two more recent studies of train workers and of teamsters have been suggestive of excess cancers in workers exposed to diesel exhaust, but require further confirmation.

To better appraise the potential lung cancer risk of exposure to diesel emissions, Cuddihy et al. (1980) attempted to compare the risks known to be associated with other carcinogenic exposures to that of diesel exposure. They assumed that the concentration of particles and of benzo(a)pyrene in urban air, in air near coke ovens, and in cigarette smoke can be used as indices of the total lung cancer risks in exposed populations. Utilizing the conclusion reached by the NAS (1980) that diesel particulates are not more carcinogenic or mutagenic than cigarette smoke or coke oven emissions on a unit particle mass basis, they applied the risk factor for respiratory cancer from these exposures to diesel emissions. Assuming that the average particle concentration inhaled by 160 million people living in urban settings was 100 $\mu g/m^3$ and that the 50 million people living in rural settings

TABLE 4.2-28

EPIDEMIOLOGIC STUDIES OF CANCER IN OCCUPATIONS EXPOSED TO DIESEL EXHAUST *

Author	Population Studied	Findings	Comments
Raffle (1957)	Males London Transportation workers 45-65 years old, 1950-1955	96 cases lung cancer. No excess lung cancer attributed to diesel exhaust exposure	inadequate duration of exposure and latency period at time of study. Diesel exhaust exposure estimated.
Kaplan (1959)	Baltimore and Ohio Railroad workers, 1953-1958	154 lung cancer deaths. SMR[a] slightly lower than national rates. No differences in rates for exposed and non-exposed workers	inadequate duration of diesel exhaust exposure and latency period at time fo study. No consideration of transefer retirement or duration of exposure
Hueper (1955)	Two large railroad companies 1939-1950	133 cases lung cancer, about 3:1 ratio observed expected in operating workers compared to non-operating workers	inadequate duration of diesel exposure at time of study. Rates not included.
Heino et al (1978)	Finish railroad workers 30-52 years old, 1959-1973	47 tumors in engineers significantly greater number than in trainmen or railroad clerks	small numbers of cases not analyzed by tumor-type
Wegman and Peters (1978)	Massachusetts tumor registry, 1965-1972	91 cases oat cell cancer excess in transportation equipment operatives	no specific for diesel exhaust exposure
Luepker and Smith (1978)	Central state teamsters May-July 1976	34 respiratory tract cancer deaths, increased SMR all age groups, significant for age 50-59	small number of cases, short period of observation

SMR[a] - Standardized Mortality Ratio

* Reprinted from Schenker, 1980. See source for complete references

were exposed to 30μg/m^3 of particulates, and assuming that 8000 m^3/yr of air was breathed by each person with 25% of the inhaled compounds deposited deeply in the lungs, they estimated that less than 30 lung cancers/yr would be expected to occur in the U.S. population after 1995 due to diesel particulates, for an individual risk of less than 1 cancer case per million/year. Some of these assumptions have been rejected by other authorities (EHL 1980) who point to the limitation of comparisons of coke oven and diesel emissions. It is noteworthy in this regard that recent studies have found that benzo(a)pyrene accounted for only up to 2% of the mutagenic activity found in air pollution samples taken in the daytime, and up to 4% of the night samples (Moller et al. 1982). From their overall data, these investigators concluded that most of the mutagenicity measured in airborne particles in daytime samples collected at street level in an area with dense traffic, indeed originated from motor vehicles (ibid).

Conclusion

At present, all that can be concluded is that although mutagenic activity has been identified in diesel exhaust, the cancer risk to human health has not yet been determined. At the present time, there is no strong human evidence to link exposure to diesel emissions to an increase in cancer, nor for that matter is there any strong human data to assess the adverse effect of diesel particulates on pulmonary function. The many variables determining the exact chemical nature of diesel particulates make any quantitative statements regarding health risk of diesel particulates as a whole very tenuous.

4.2.6 Vapour-Phase Hydrocarbons

It is generally agreed that hydrocarbons as a class do not present a direct health hazard, although they must be controlled on the basis of their contribution to photochemical smog. As discussed in Chapter 3, the U.S. nonmethane hydrocarbon air quality standard of 160 $\mu g/m^3$ (0.24 ppm) maximum 3-year average concentration, not to be exceeded more than once annually, had been set by the EPA in 1970 to control the ambient levels of specific smog components as stipulated by the oxidant standard at that time. Ambient air quality standards for hydrocarbons as a whole have not been set in other jurisdictions, and have since been abandoned in the U.S.

In October 1981 the EPA reviewed their air quality criteria for vapour phase hydrocarbons. The document discussed the characteristics of the various groups of hydrocarbons, reviewed the criteria developed in 1970 for the standard-setting process, and examined the data on hydrocarbons that had accumulated over the subsequent decade. Both the 1970 as well as 1981 documents covered only those organic compounds composed solely of carbon and hydrogen and that occur in the atmosphere in the gaseous phase. It specifically excluded substituted organics, such as the halogenated hydrocarbons, as well as organic compounds that occur as aerosols or particulates. Separate studies of polycyclic organic matter have also been conducted (EPA,Feb. 1979). These compounds are associated with suspended particulates, principally respirable particles, and as discussed in Section 4.2.5, the major health concern is their carcinogenicity.

Aliphatic and Alicyclic Hydrocarbons: In the case of the aliphatic hydrocarbons as a group, the EPA (1981) noted that their tolerable concentration in air is limited only by the percent of available oxygen (18%). It was pointed out, however, that pentane, hexane, heptane and octane must be controlled more tightly based on their potential to cause chronic neurological disorders. The concentration of 350 $\mu g/m^3$ (120,100,85 and 75 ppm respectively) was designated as their Threshold Limit Valve (NIOSH, 1977) for occupational exposures. It was also noted that the aliphatics emitted as gases in the atmosphere have not been implicated as carcinogenic.

With respect to the alicyclics (naphthenes), the EPA concluded that they act as central nervous system depressants with a relatively low order of acute toxicity. Further, it was noted that cumulative toxicity from repeated exposure to low concentrations is unlikely, as the alicyclics do not tend to accumulate in body tissues.

Aromatic Hydrocarbons: It is known that the aromatic hydrocarbons are more irritating to the mucous membranes than equivalent concentrations of the aliphatics or alicyclics, but that systemic injury is still unlikely at anything near ambient levels. The EPA did, however, single out benzene as present in ambient air at levels representing a risk for leukemia, pancytopenia and chromosomal aberrations (NRC, 1976; Snyder and Kocsis, 1975; and EPA, Sept. 1978). It was noted that haematological abnormalities have developed in humans as a result of repeated exposure to benzene concentrations ranging down to 336 and 192 mg/m^3 (105 and 60 ppm) (Hardy and Ellans, 1948; Wilson, 1942), and that Pagnatto _et al_. have suggested that haematological changes have occurred in rubber-coating plants at benzene concentrations as low as 64 to 80 mg/m^3 (20 to 25 ppm). The studies showing the chromosomal aberrations can result from chronic exposure to benzene at concentrations ranging from 80-480 mg/m^3 (25-150 ppm) were noted, as was one study in which significant effects were noted at 2-3 ppm time-weighted average (Blaney, 1950). The fact that the National Institute on Occupational Safety and Health had recommended the stringent standard of 0.3 mg/m^3 (1 ppm) benzene in air as an 8-hour average with a ceiling level of 16 mg/m^3 (5 ppm) for any 15-minute period during this 8-hour day was also alluded to. Other reports were referred to (EPA, Sept. 1978; EPA, 1978) in which it was estimated from studies by Infante _et al_. (1977), Otto _et al_. (1977) and Askoy _et al_. (1974, 1976, 1977) that 90 cases of leukemia per year could be attributed to the current low ambient level of benzene (about 1 ppb or 0.3 $\mu g/m^3$) to which the population at large is generally exposed. (This represents 0.23-1.62% of total leukemia deaths in the U.S. based upon 1973 statistics). With respect to exposure to toluene, xylene and trimethyl benzenes which are generally less toxic and volatile than benzene, it was noted that thorough reviews (e.g. NIOSH, 1973; Cohn,

1979) have concluded that 100 ppm is an adequate threshold for occupational exposure. This concentration of toluene was set to prevent CNS-depressant effects, although it was noted that exercise might modify the situation, necessitating a lower standard. The 100 ppm (435 mg/m^3) standard for xylene was designed to prevent irritating and narcotizing effects from occurring. Data regarding chronic effects have been inconsistent.

Hydrocarbon Mixtures: The EPA noted that acute exposure to high concentrations of hydrocarbon mixtures in gasoline vapours can sensitize the myocardium (heart musculе) such that sudden death from ventricular fibrillation may occur (Ainsworth, 1961; Wang and Irons, 1961; Aaidin, 1958; Nelms et al.,1970; Poklis, 1976; Chenoweth, 1946). Acute exposure to lower concentrations has been noted to cause eye irritation and nonspecific anesthetic or narcotic effects. Slight dizziness and irritation of eyes, nose and throat had been reported to occur at 1-h exposure to 480 - 810 mg/m^3 (160 - 270 ppm) (Drinker et al., 1943). A later study noted no symptoms at exposures of up to 3000 mg/m^3 (1000 ppm) (Davis et al., 1960). Chronic exposure to gasoline is thought also to produce non-specific symptoms such as fatigue, muscle weakness, nausea, vomiting, abdominal pain and weight loss, as well as central and peripheral neurological effects (Sinyard, 1970). Assuming an additive effect of benzene-induced damage with that attributable to other hydrocarbons (ACGIH, 1976), the TLV for gasoline was set at a 900 mg/m^3 (300 ppm) Time-Weighted Average (TWA) over an 8-hr period with a 15 minute ceiling of 3000 mg/m^3 (1000 ppm) (McDermott and Killiny, 1976). Utilizing the TLV for benzene of 0.3 mg/m^3 (1 ppm) (which was overturned by the Supreme Court) the TLV for gasoline would have been 450 mg/m^3 (150 ppm). The lowest standard for a miscellaneous hydrocarbon mixture was suggested to be 90 ppm based upon a series of 17 inhalation studies with animals in lethal atmosphere or studies of the sensory response of human subjects (Carpenter et al., 1975-1977). The EPA concluded that this constituted additional evidence for the low level of toxicity of hydrocarbons, with the exception of benzene.

Aldehydes, etc: The irritating effect of aldeydes and peroxyacylnitrates on mucous membranes of the eyes, nose and throat has been well-documented. The EPA(1981) has noted that these compounds are responsible for the only health effects reported from exposure to smog products derived directly from hydrocarbons (as opposed to indirectly derived products such as ozone), at levels approximating those found in the ambient air. Acrolein and formaldehyde were identified by the EPA (1981) as the most effective irritants of the aldehydes, while acetyl and benzoyl derivatives were designated the most potent from the peroxyacylnitrates.

The EPA recommended that exposure to aldehydes be reconsidered when the National Academy of Sciences had completed its report. Meanwhile no ambient air quality standard has been set for formaldehyde or any of the other aldehydes or peroxyacylnitrates, although there seems to be growing concern especially with respect to the sensitization potential of formaldehyde. It has not been possible, in fact, to determine an exposure level at which some hypersensitive individuals will not respond. Allergic contact dematitis has been documented in some individuals exposed to formaldehyde vapour, but of course, the asthmatic response has been the focus of the concern. With respect to the effects of chronic exposure to low doses of formaldehyde some data regarding the effect on the respiratory system has been reported. It has not been established whether or not sufficient formaldehyde is absorbed to produce systemic effects, and ongoing research is examining its potential mutagenic and carcinogenic potential.

Conclusion: It seems that there is little cause for concern about health effects attributable directly to hydrocarbons as a class, at levels in the range currently found in the ambient air. There is evidence that low levels of exposure to benzene are associated with increased risk of leukemia, aplastic anaemia and other blood system disorders. There is also some basis for concern regarding ambient exposure to aldehydes, however, reasonable quantitative evaluations cannot be made at this time.

4.2.7 Acid Precipitation

As explained in Chapter 2, several of the pollutants associated with automobile exhaust have been implicated as precursors to acid deposition. Data related to health effects of acidic deposition have been thoroughly reviewed by the Impact Assessment Work Group for the Memorandum of Intent on Transboundary Air Pollution (MOI, 1983). The Workgroup concluded that while there was little cause for concern over direct health effects from acidic deposition, the potential for indirect health effects associated with acid precipitation merits consideration. These effects include: (1) contamination of the food chain by metallic substances, especially mercury; (2) leaching of watersheds and corrosion of storage and distribution systems, leading to elevated levels of toxic substances; and (3) health implications of recreational activities in acidified water. The nature of each of these concerns will be described briefly.

Contamination of Edible Fish: Several studies providing evidence that acidic deposition may alter the biogeochemical cycle of metals were alluded to by the workgroup. It was clearly pointed out that a correlation existed between low pH in lakes and higher mercury concentrations in some species of fish. Various theories as to the mechanisms of this phenomenon were put forward. It was concluded that although the extent to which acid deposition may have contributed to mobilization and retention of mercury in fish is speculative, fish harvested from these lakes present a potential health hazard to humans. Epidemiological studies conducted in Canada which investigate the health of populations, especially natives, that were exposed to high concentrations of mercury in food were referred to. For example, the study by Rudy (1980) which documented neurological abnormalities in Cree males in association with mercury exposure was cited. Although there is no definitive data at present regarding the role of acid precipitation careful consideration to the potential health risk of long-term exposure to elevated levels of metals, particularly mercury in food, was advocated.

Contamination of Drinking Water: It was noted by the Work Group (MOI,1983) that acidic deposition can increase the concentration of toxic metals in drinking water by: (1) increasing the deposition of metal in soluble forms (e.g. mercury); (2) leaching of metals from the watershed and from sediments; and (3) acid corrosion of materials used in reservoirs, drinking water distribution systems and cisterns. While no clear evidence of health effects arising from the consumption of drinking water contaminated with metals due to acidic deposition are reported in the literature, some potential problems are identified. In New York State, for example, water from the Hinkley reservoir had become acidified to such an extent that lead concentrations in drinking water at the tap exceeded the maximum levels for human use (50 μg/L) recommended by the New York State Department of Health (Turk and Peters, 1978). Moreover, lead levels in tap water from cisterns were found to be much higher than those found in the source water (Sharpe et al. 1980), thought to be due to acid corrosion of the lead soldered joints in the cistern and plumbing. Thus the Work Group concluded that cistern water users are at special risk in areas of high acidic depostion, although no adverse health effects resulting from consumption of such water have been reported.

Recreational Activities in Acidified Water: Some concern has been expressed that recreational activities in affected waters (e.g. swimming) may prove to be detrimental to human health. It is felt that the eye would be the most likely to be affected, however, to date, no compelling evidence exists to suggest that ocular clinical effects are indeed produced by exposure to acidic waters. Research in this area is continuing.

Summary: While there is considerable evidence that the precursors to acidic deposition may have serious adverse effects on health, there is no evidence of any deleterious health effects from acid deposition itself. Some preliminary evidence suggests that acid precipitation may indirectly affect human health. Particularly sensitive areas include those with poorly buffered lakes and streams (with viable fish populations), watersheds with unusual accumulations of metals and areas which lack drinking water treatment facilities or which have substantial lead plumbing. Populations at greatest risk include those dependent on fish from acidified waters as a major dietary staple, those with elevated mercury or lead blood levels from other exposures, those dependent on cisterns as a primary source of drinking water, and women of childbearing age as well as children (MOI, 1983).

4.2.8 Summary

The possible health effects of the various components of automotive emissions are summarized in Table 4.2-29 along with an approximation of the exposure levels at which these effects have been demonstrated or may be expected to occur.

It can be seen from this table that carbon monoxide (CO) has a deleterious effect on cardiac function which is of greatest relevance to individuals with cardiovascular disease. Carbon monoxide also has an adverse effect on work performance and on central nervous system functions. Impairment of vigilance or performance of complex tasks can occur at low levels of CO exposure which may be of significance to the general population, especially with respect to its possible effect on driving skills. No such effect has, however, been clearly documented. Pregnant women, their fetuses and newborn babies, are at increased risk of carbon monoxide toxicity, but precise quantification of risk in this group is not yet possible.

Exposure to nitrogen dioxide (NO_2) affects sensory perception and causes irritation to the mucous membranes of the respiratory tract. The adverse effect of NO_2 on lung function may be of particular relevance to asthmatics. It also appears that NO_2 increases susceptibility to infection, possibly accounting for an increased incidence of respiratory disease, most notably in children.

Exposure to ozone may induce abnormalities in various organs throughout the body. Again, the respiratory system is the site of greatest damage. Ozone impairs lung function, with the magnitude of the effect dependent on a variety of factors, including individual susceptibility and level of exercise. Decreased performance, exacerbation of asthma and increased susceptibility to infection have also been linked to ozone exposure.

Diesel exhaust is known to contain a high concentration of particulates, with the latter having been linked to increased mortality among elderly

TABLE 4.2-29

SUMMARY OF POSSIBLE MAJOR EFFECTS OF AUTOMOTIVE EMISSIONS ON HEALTH

Substance	Effect	Susceptible Population	Exposure*	For further information regarding Dose-Response see:
Carbon Monoxide	Deleterious effect on cardiac function	People with cardiovascular disease	COHb of 2%**	Table 4.2-1
	. Aggravation of angina or peripheral vascular disease		COHb of 2.5-3%	Tables 4.2-2, 4.2-3 Figures 4.2-2, 4.2-3
	. Increased risk of cardiac death		COHb of 2-3%	Tables 4.2-4, Figure 4.2-4
	Decreased work capacity	General population	COHb of >4%	Figure 4.2-5
	Decreased vigilance	Healthy young adults	COHb of 3-4%	Table 4.2-5
	Deleterious effect on fetal development	Pregnant women and newborns	Fetal COHb of 7%, maternal COHb of >4%	Tables 4.2-6, 4.2-7
Nitrogen Dioxide	Adverse effect on lung function	Healthy adults	4700 $\mu g/m^3$ (2.5 ppm)	Tables 4.2-14, 4.2-15 Figure 4.2-8
		Asthmatics***	940 $\mu g/m^3$ (0.5 ppm)	Tables 4.2-15, 4.2-19, Figure 4.2-8
	. Increased sensitivity to bronchoconstrictors		94-188 $\mu g/m^3$ (0.05-0.1 ppm)	Tables 4.2-14, 4.2-21
	. Increased incidence of respiratory symptoms	Young children	752-1880 $\mu g/m^3$ (0.4-1 ppm)	Tables 4.2-17, 4.2-18, 4.2-21

TABLE 4.2-29 (CONT'D)

Substance	Effect	Susceptible Population	Exposure*	For further information regarding Dose-Response see:
Ozone	Adverse effect on lung function	Healthy adults		
	. With light exercise		740 μg/m^3 (0.37 ppm)	Table 4.2-23
	. With vigorous exercise		300 μg/m^3 (0.15 ppm)	Table 4.2-23
		Sensitive population***	300 μg/m^3 (0.15 ppm)	Tables 4.2-23, 4.2-25, Figure 4.2-10
	Chest discomfort and irritation of eyes and respiratory tract	General population	300-500 μg/m^3 (0.15-0.25 ppm)	Tables 4.2-23, 4.2-25
	Aggravation of respiratory disease	People with asthma, chronic bronchitis, or emphysema	300 μg/m^3 (0.15 ppm)	Tables 4.2-24, 4.2-25 Figure 4.2-10
	. Reduced resistance to bacterial infection	Sensitive population***	360 μg/m^3 (0.18 ppm)	Table 4.2-25
	Decreased performance	Healthy adults	200-300 μg/m^3 (0.10-0.15 ppm)	Table 4.2-24
Diesel Particulates	Increased mortality (cardio-respiratory)	Elderly individuals and people with respiratory disease	500 μg/m^3 TSP	Tables 4.2-26 4.2-27 Figure 4.2-26
	Increased morbidity	As above	250 μg/m^3 TSP	Tables 4.2-26 4.2-27 Figure 4.2-26
		Asthmatic children	150 μg/m^3 TSP 100 μg/m^3 Chronic exposure	Tables 4.2-26 4.2-27

TABLE 4.2-29 (CONT'D)

Substance	Effect	Susceptible Population	Exposure*	For further information regarding Dose-Response see:
	Increased risk of cancer	General population		Table 4.2-28
Vapour-Phase Hydrocarbons	--			
Benzene	Increased risk of leukemia and other blood disorders	General population	300 μg/m^3 (1 ppm)	--
Acid Precipitation	Increased risk of mercury poisoning	Population dependent on fish		--
	Increased risk of lead poisoning and poisoning by other heavy metals	Population with poor drinking water treatment facilities or with lead plumbing		--
	Possible eye irritation	Recreational swimmers, etc.		--

* These exposure levels represent the minimum concentration reported to be associated with the listed effect. The duration of exposure to this concentration necessary to produce the effect noted is indicated in the tables listed in the final column.

** The level of carboxyhaemoglobin (COHb) associated with deleterious effect on cardiac function was reported to be even lower than 2% by one expert. The concentrations and duration of exposure to carbon monoxide which produce this level of COHb are described in Tables 4.2-9 and 4.2-10.

*** People with hypersensitive airways are at increased risk from certain exposures. Additional data regarding susceptible populations are provided in the appropriate subsections.

individuals and patients with respiratory disease (chronic bronchitis, emphysema, etc). Exposure to particulates is also known to cause exacerbation of disease in such patients, and studies have linked the frequency of asthma attacks and incidence of acute respiratory disease in children with exposure to particulates. The major health concern regarding diesel emissions is, however, its potential to be cancer-causing. At present, no convincing human evidence exists to substantiate, let alone quantify, this risk.

Hydrocarbons as a class provide little direct health effect at ambient levels. Some hydrocarbons, however, can be hazardous at levels approximating those found in the ambient air. Benzene, for example, known to be capable of producing serious blood disorders, including leukemia, may be of concern in some specific settings. Aldehydes, such as formaldehyde, which may be formed from hydrocarbons emitted in automobile exhaust, may be irritating to the mucous membranes or cause asthmatic attacks in sensitive individuals. Quantification of this effect is not possible at this time.

Acid precipitation may also indirectly affect health by increasing the risk of mercury poisoning, lead poisoning or intoxication from other elements in some areas and populations.

This chapter has illustrated the many controversies that exist regarding the lowest level of exposure which may be harmful to human health. As concluded by the National Academy of Sciences in 1974, "in no case is there evidence that threshold levels have clear physiological meaning, in the sense that there are genuine adverse health effects at above some level of pollution, but no effects at all below that level. On the contrary, evidence indicates that the amount of health damage varies with the upward and downward variations in the concentration of the pollutant, with no sharp lower limit". (EPA, June 1978). Therefore, as discussed in Chapter 3, a cautious approach must be advocated in the process of standard setting.

4.3 Terrestrial Environment

4.3.1 Introduction

This section describes the effects of nitrogen oxides, photochemical oxidants, and acid precipitation upon the terrestrial environment. For the purposes of this evaluation, we have restricted the discussion to effects upon vegetation communities, including natural ecosystems, agricultural crops, forests, and ornamental plants. No attempt has been made at this time to assess indirect effects upon animal communities due to emission effects on vegetation.

In any study of this nature, it is important to determine what is an "effect" on vegetation, and when is it to be considered adverse. This is by no means an easy task. All identifiable plant responses, such as reductions in photosynthetic rates, leaf necrosis, yield reductions, etc., may not necessarily be considered adverse under all circumstances (OAQPS, 1982).

The vegetation effects or responses which will be considered in this section generally fall into three areas; foliar injury, growth and yield reduction, and physiological or biochemical changes. Dose/response studies have generally examined such responses and the data reported in terms of effects in these categories.

The experimental evaluation of pollutant exposure responses has proven very challenging, due to difficulties in extrapolating from greenhouse or laboratory situations to the varying conditions found in urban and rural environments. Exposure systems used in air pollution research on vegetation have been reviewed and evaluated by Heagle and Philbeck (1979) and the U.S. Environmental Protection Agency (1982a). These include:

a) laboratory systems varying in complexity from simple bubblers to environmental chambers to continuous stirred tank reactors to study pollutant uptake (Rogers et al., 1977).

b) greenhouse exposure systems designed to provide uniform pollutant concentrations with minimal environmental alteration (Heck et al., 1968).

c) field exposure systems attempt to minimize deviations from the ambient environment and replicate as closely as possible the conditions characteristic of agriculture or natural ecosystems. These range from extrapolations of greenhouse and laboratory designs to the use of chemical protectants. Many innovative designs have evolved. These include:

  - field chamber systems such as the open-top chamber systems described by Heagle et al., (1973) and Mandl (1976) which are the most popular field exposure designs. Other designs are fully enclosed by film (Thompson and Taylor, 1966; Oshima, 1978). These chamber systems combine controlled exposure capabilities with placement in the ambient environment with respect to other variables.

  - field exposure systems without chambers combine the advantage of exposure of plants to pollutants under ambient-like conditions, balanced against the loss of some control over fumigant level and nature of the exposure, and sensitivity to wind conditions. Examples include the zonal air pollution system (ZAPS) (Lee et al., 1978), and the linear gradient system (Reich et al., 1980; Laurence et al., 1982). Modifications of ZAPS have been described as possibly the best systems for exposure of perennial or tree crops (U.S. EPA, 1982a).

d) gas exchange systems for field use have been described by Bingham and Coyne (1977) and Legge et al., (1979) which are capable of controlling temperature and humidity while measuring leaf gas-exchange parameters, although not being exposure systems in the truest sense.

Exposure to pollutants in the natural environment is influenced by source strength, meteorological factors and topography, so that periods of significant pollution, termed episodes, are periodic, vary in duration, and often occur several times during the growth of plants. It is not yet entirely clear which components of an exposure are most important in producing vegetative responses (U.S. EPA, 1982a) so that experimental characterization of exposures is attempting to simulate realistic but controlled conditions so as to better understand the mechanisms and extent of plant sensitivity.

Work to date, however, suggests that there is no stable relationship between the concentration of pollutant in the air and the plant's internal flux so that it is not now possible to relate internal flux, which directly affects plant metabolism, to an exposure statistic that can be aggregated to represent seasonal exposures. As well, the results of short-term exposure experiments cannot effectively be extrapolated to evaluate its significance in relation to long-term ambient exposures (U.S. EPA, 1982a). Dose (concentration x time) statistics cannot therefore be aggregated to represent long-term exposures.

4.3.2 Nitrogen Oxides

Effects on Vegetation

The effects of nitrogen oxides (NO_x) on terrestrial ecosystems have recently been reviewed in detail by the United States Environmental Protection Agency for the preparation of air quality criteria and standards (U.S. EPA, 1982b; OAQPS, 1982). As well, Mansfield and Freer-Smith (1981) have recently emphasized European findings in a review of the effects of NO_x and other pollutants on urban plant growth. These authors note that much of the earlier literature is of only limited value since it tended to be dominated by experiments that employed levels of NO_x higher than those encountered in even the most polluted urban environments.

Natural Ecosystems: At the present time, there are insufficient data to accurately determine the impact of NO_x on terrestrial plant, animal, or microbial communities. Evidence of visible injury to plant communities has seldom been demonstrated and may in fact represent only a fraction of the actual harm done to terrestrial communities (U.S. EPA, 1982b). Work by Hepting (1964) has suggested that the vigour and survival rates of plants have been affected by air pollution, but because of complicating factors in the wild, only severe injury could likely be ascribed to a particular episode.

Existing studies of the effects of air pollution on ecosystems and plant communities by Parmeter and Cobb (1972), Wenger et al. (1971) and others have concluded only that further research on the influence of NO_x on plant communities is required. The U.S. EPA (1982b) has concluded that the available information is too small to warrant meaningful generalizations at this time, but has assembled data detailing experimental findings on NO_x effects on individual plant species, including some forest trees. These are presented in Table 4.3-1 in terms of relative sensitivity to nitrogen dioxide.

The three relative sensitivity categories in Table 4.3-1 are described by the compilers (EPA 1982b) as approximate, because they are based upon subjective criteria obtained from several sources. Thus, no generalizations about concentrations or durations of exposure are applicable.

Of the coniferous tree species cited, larches are described as susceptible, while knee pine, Austrian pine and English yew were considered tolerant. Intermediate in sensitivity were white fir, Japanese fir, silver fir, white spruce, and Colorado blue spruce. Susceptible deciduous species were white birch and brittlewood, and sensitive "weeds" included mustard and common sunflower.

Little attention has been given to the effects of NO_x on animal populations and natural communities and conclusions cannot be developed based on the very sparse data. Similarly, no data regarding ambient NO_x concentration effects on microbial processes or populations are available. The National Academy of Sciences (1977) and U.S. EPA (1982b) concluded that ambient NO_x concentrations probably do not significantly affect biologic processes in natural ecosystems, but were unable to support this view with experimental data.

TABLE 4.3-1 RELATIVE SENSITIVITY OF SEVERAL PLANT SPECIES TO NITROGEN DIOXIDE (HECK AND TINGEY, 1979; MACLEAN, 1977; TAYLOR AND MACLEAN, 1970; TAYLOR ET AL., 1975; U.S. ENVIRONMENTAL PROTECTION AGENCY, 1971)

Plant Type	Susceptible	Intermediate	Tolerant
Coniferous Trees	Larix decidua Mill. (European Larch) Larix leptolepis Gord. (Japanese larch)	Abies alba Mill. (White fir) Abies homolepis Sieb. & Zucc. (Nikko or Japanese fir) Abies pectinata DC (Common Silver Fir) Chamaecyparis Lawsoniana [Murr.] Parl (Lawson's cypress) Picea glauca [Moench] Voss (White Spruce) Picea pungens glauca, Regel (Colorado Blue Spruce)	Pinus Mugo Turra (Knee pine or dwarf mountain pine) Pinus nigra Arnold (Austrian pine) Taxus baccata L. (English yew)
Field Crops & Grasses	Avena sativa L. (Oats) cv. Clintland 64 cv. 329-80 cv. Pendek Bromus inermis, L. (Bromegrass) cv. Sac Smooth Hordeum distichon L. (Barley) Medicago sativa, L. (Alfalfa) Nicotiana glutinosa L. (Tobacco) Nicotiana tabacum L. (Tobacco) Scorzonera hispanica L. (Viper's grass) T. incarnatum L. (Crimson or Italian Clover) Trifolium pratense L. (Red clover) Triticum vulgare, Vill. (Wheat) cv. Wells Vicia sativa L. (Spring vetch)	Gossypium hirsutum, L. (Cotton) cv. Acala 4-42 cv. Paymaster Nicotiana tabacum, L. (Tobacco) cv. White Gold cv. Bel-B cv. Bel W3 Poa annua, L. (Annual bluegrass) Secale cereale L. (Rye) Triticum aestivum L. (Wheat) Zea Mays L. (Sweet Corn)	Nicotiana tabacum, L. (Tobacco) cv. Burley 21 Poa pratensis L. (Kentucky bluegrass) Sorghum sp. (Sorghum) cv. Martin Zea Mays L. (Corn) cv. Pioneer 509-W cv. Golden Cross
Fruit Trees	Malus sp. (Showy apple) Malus sylvestris Mill. (Apple) Pyrus communis L. (Wild Pear)	Citrus sp. (Orange, grapefruit, tangelo) Citrus sinensis (L.) Osbeck (Navel Orange)	Hosta plantaginea (Lam.) Aschers (Fragrant plantain lily)
Garden Crops	Allium porrum L. (Leek) Apium graveolens L. (Celery) Brassica oleracea botrytis, L. (Broccoli) cv. Calabrese Daucus carota L. (Carrot) Lactuca sativa, L. (Lettuce) Petroselinum hortense Hym. (Parsley) Phaseolus vulgaris, L. (Bean) cv. Pinto Pisum sativum L. (Pea) Raphanus sativus L. (Radish) cv. Cherry Belle Rheum rhaponticum L. (Rhubarb) Sinapis alba (White mustard)	Apium graveolens rapaceum (Celery) Cichorium Endivia, L. (Endive) Ruffee Fragaria chiloensis grandiflora (Pine strawberry) Lycopersicon esculentum, Mill (Tomato) cv. Roma Phaseolus vulgaris humilis Alef. (Bush bean) Solanum tuberosum L. (Potato)	Allium cepa L. (Onion) Asparagus officinalis L. (Asparagus) Brassica caulorapa Pasq. (Kohlrabi) Brassica oleracea acephala DC (Kale) Brassica oleracea capitata L. (Cabbage) Brassica oleracea capitata rubra L. (Red cabbage) Cucumis sativus, L. (Cucumber) cv. Long Marketeer Phaseolus vulgaris, L. (Bush Bean)
Ornamental Shrubs and Flowers	Antirrhinum majus L. (Giant Snapdragon) Begonia multiflora (Tuberous-rooted begonia) Begonia rex, Putz. (Begonia) cv. Thousand Wonders White Bougainvillea spectabilis Willd (Bougainvillea) Callistephus chinensis (L.) Nees (China aster) Chrysanthemum sp. (Chrysanthemum) cv. Oregon Hibiscus Rosa-sinensis L. (Chinese hibiscus) Impatiens sultani, Hook (Sultana) cv. White Imp Lathyrus odoratus L. (Sweet pea) Lupinus angustifolius L. (Lupine) Nerium oleander L. (Oleander) Pyracantha coccinea Roem. (Fire thorn) Rhododendron canescens [Michx.] Sweet (Hoary Azalea) Rosa sp. (Rosa) Vinca minor L. (Periwinkle) cv. Bright Eyes	Dahlia variabilis Willd. (Dahlia) Fuchsia hybrida Voss (Fuchsia) Gardenia jasminoides Ellis (Cape Jasmine) Gardenia radicans Thunb. (Gardenia) Ixora coccinea L. (Ixora) Ligustrum lucidum Ait. (Ligustrum) Petunia X hybrida Hort. Vilm.-Andr. (Common Garden Petunia) Pittosporum tobira Ait. (Japanese pittosporum) Rhododendron catawbiense Michx. (Catawba rhododendron)	Carissa carandas L. (Carissa) Codiaeum variegatum Blume (Croton) Chrysanthemum leucanthemum L. (Daisy) Convallari majalis L. (Lily-of-the-valley) Erica carnea L. (Spring heath) Gladiolus communis L. (Gladiolus) Erica sp. (Heath) Hosta sp. (Plantain lily) Juniperus conferta Parl. (Shore juniper) Rhododendron sp. (Alaska)
Trees & Shrubs	Betula pendula Roth. (European white birch) Melaleuca leucadendra (L.) L. (Brittlewood)	Acer platanoides L. (Norway maple) Acer palmatum Thunb. (Japanese maple) Tilia grandiflora (Summer) Tilia cordata Mill. (Small-leaved European linden)	Carpinus betulus L. (European hornbeam) Fagus sylvatica L. (Beech) Fagus sylvatica atropurpurea Kirchn. (Purple-leaved beech) Ginkgo biloba L. (Gingko) Quercus robur L. (English oak) Robinia pseudoacacia L. (Black locust) Sambucus nigra L. (European elder) Ulmus glabra Huds. (Scotch elm) Ulmus montana With. (Mountain elm)
Weeds	Brassica sp. (Mustard) Helianthus annuus L. (Common Sunflower)	Malva parviflora L. (Cheeseweed) Stellaria media (L.) Cyrill (Chickweed) Taraxacum officinale Weber (Dandelion)	Amaranthus retroflexus L. (Pigweed) Chenopodium album L. (Lamb's-quarters) Chenopodium sp. (Nettle-leaved goosefoot)

Source: EPA 1982b

Agriculture: A greater amount of information is available related to NO_x effects on agricultural crops. The mode of action depends upon NO_2 uptake into the leaves since perturbations occur at cellular sites within mesophyll tissue (U.S. EPA, 1982b). NO_2 uptake in corn (Zea mays) and soybean (Glycine max) was found by Rogers et al. (1979) to be directly related to stomatal resistance in the range of 0 to 1.09 mg/m^3 (0-0.58 ppm), and that NO_2 uptake increased with light intensity through the action of light on stomatal resistance. Other studies have related NO_2 uptake to internal leaf factors (Srivastava et al., 1975), who also noted that uptake rate increased with concentration and declined with increasing exposure time.

Observed effects of NO_x on vegetation fall into three principal categories; foliar injury, growth and yield reductions, and physiological and biochemical changes (OAQPS, 1982). Acute exposures to NO_2 typically elicit different responses than do chronic exposures. Leaf necrosis, expressed as light brown, irregularly shaped necrotic lesions usually at or near the tips of leaves, usually characterizes acute exposure responses, with the affected area varying with the magnitude of the exposures. Long exposures to low NO_2 concentrations often result in leaf chlorosis, where greenish yellow spots or yellowing of the leaf surface occur

Heck and Tingey (1979) reported results from a series of short-term (1 hour) exposures to NO_2 at 15.0 mg/m^3 (8 ppm), 30.1 mg/m^3 (16 ppm) and 60.2 mg/m^3 (32 ppm) (Table 4.3-2). Only brome grass (Bromus inermis) and tomato (Lycopersicon esculentum) showed foliar injury at 15.0 mg/m^3 (8 ppm), whereas all species tested showed visible injury at 60.2 mg/m^3 (32 ppm). A second experiment varied both time and concentrations, varying from 0.5 to 7 hours and 3.8 to 37.6 mg/m^3 (2 to 20 ppm) NO_2 (Table 4.3-3). An important conclusion (U.S. EPA, 1982b) was that the extent of injury was greatest when NO_2 levels were high, even for short time periods. Therefore, dose alone is not always a good prediction of injury.

TABLE 4.3-2 ACUTE INJURY TO SELECTED CROPS AFTER A 1-HOUR EXPOSURE TO NITROGEN DIOXIDE (HECK AND TINGEY, 1979)

Plants (Common, Cultivar, Scientific)[d]	Injury Index *		
	8 ppm	16 ppm	32 ppm
Tomato, Roma[b] (*Lycopersicon esculentum*)	1	48	100
Wheat, Wells[c] (*Triticum durum*)	0	47	90
Soybean, Scott[b] (*Glycine max*)	0	26	100
Tobacco, Bel W_3[b] (*Nicotiana tabacum*)	0	23	97
Bromegrass, Sac Smooth[b] (*Bromus inermis*)	2	17	97
Swiss Chard, Fordhook Giant[c] (*Beta vulgaris cicla*)	0	11	62
Tobacco, White Gold[b] (*Nicotiana tabacum*)	0	1	70
Cotton, Acala 4-42[c] (*Gossypium hirsutum*)	0	0	54
Beet, Perfected Detroit[c] (*Beta vulgaris*)	0	0	36
Orchard Grass, Potomac[c] (*Dactylis glomerata*)	0	1	18
Tobacco, Bel W_3[c]	0	0	5

[a] Plants were exposed in Cincinnati, Ohio.

[b] Plants were exposed in August with light intensity at 2200 ft-c, temperature 28°C, humidity 75 percent.

[c] Plants were exposed in January with light intensity at 1400 ft-c, temperature 21°C, humidity 70 percent.

[d] Scientific name is given only when plant is first listed.

* Injury Index measured as mean percentage of the leaf area showing NO_2 injury.

Source: EPA 1982b

TABLE 4.3-3 PERCENT LEAF AREA INJURED BY DESIGNATED DOSAGE OF NITROGEN DIOXIDE (HECK AND TINGEY, 1979)

Plants (Common, Cultivar, Scientific)	Dosage (ppm x hr)	2.5	4	6	10	14	15	20	20	35
	(ppm)	5	4	3	20	7	15	5	10	5
	(hr)	0.5	1	2	0.5	2	1	4	2	7
Oats, Clintland 64 (*Avena sativa*)		0	0	0	80	2	84	0	39	21
Radish, Cherry Belle (*Raphanus sativus*)		0	0	0	95	0	90	1	31	2
Bromegrass, Sac Smooth		0	0	0	69	0	50	1	26	0
Begonia, Thousand Wonders* White[d], (*Begonia Rex*)		0	1	0	26	0	35	4	49	5
Chrysanthemum, Oregon[d] * (*Chrysanthemum* sp.)		1	1	1	34	0	41	4	25	1
Sultana, White Imp[d] * (*Impatiens sultani*)		0	0	0	51	0	26	0	24	0
Oats, 329-80[b] (*Avena sativa*)		2	2	1	32	1	18	9	14	14
Cotton, Paymaster (*Gossypium hirsutum*)		0	0	6	50	0	27	2	2	1
Wheat, Wells		3	2	1	31	3	34	3	2	1
Cotton, Acala 4-42		0	0	0	28	0	28	0	1	1
Periwinkle, Bright Eyes[d] * (*Vinca minor*)		0	0	0	13	0	20	1	23	1
Oats, Pendek[c] (*Avena sativa*)		1	2	0	39	0	2	1	2	2
Broccoli, Calabreese (*Brassica oleracea botrytis*)		0	0	0	19	0	21	0	0	0
Tobacco, Bel B (*Nicotiana tabacum*)		0	0	3	18	0	17	0	0	0
Tobacco, White Gold		0	0	1	18	0	6	0	0	0
Tobacco, Bel W_3		0	0	6	15	0	2	0	0	0
Tobacco, Burley 21 (*Nicotiana tabacum*)		0	0	0	8	0	0	0	0	0
Corn, Pioneer 509-W (*Zea mays*)		1	0	0	1	0	1	0	0	0
Corn, Golden Cross (*Zea mays*)		0	0	0	0	0	0	0	0	2
Azalea, Alaska * (*Rhododendron*, sp.)[d]		0	0	0	0	0	1	0	0	0
Sorghum, Martin (*Sorghum*, sp.)		0	0	0	0	0	0	0	0	0
Cucumber, Long Marketer (*Cucumis sativus*)		0	0	0	0	0	0	0	0	0

[a] Plants were exposed in Cincinnati, Ohio. Each value is the average of 4 replicate plants except as noted. Plants are listed in general order of sensitivity.

Injury estimates based on the average of the three most sensitive leaves except for plants indicated (*) when the estimate was based on the total leaves per plant.

Source: EPA 1982b

The foliar injury data were summarized by Heck and Tingey (1979) to develop a predictive model for NO_2 acute exposures. As reported by the U.S. EPA (1982b), the relationship is as follows:

$$C = Ao + A_1 I + A_2 T^{-1}$$

where C = concentration (ppm)

Ao, A_1, A_2 - constants (partial regression coefficients) for pollutant, plant species and environmental conditions.

I - percent injury

T = time (hours)

In this fashion, the separate roles of time and concentration are recognized, permitting the development of three-dimensional injury response surfaces. Table 4.3-4 presents projected exposure concentration and duration estimates required to produce foliar injury on susceptible, intermediate, and tolerant plants at the threshold injury level.

Visible leaf injury is the most readily detectable and frequently reported symptom of exposure, and for this reason, has commonly been used in attempts to report damage to economic crops (OAQPS, 1982). Foliar injury may occur without detectable decreases in growth or yield, and conversely, such decreases may in fact occur without visible symptoms.

Because of the importance of exposure time in NO_2 injury to plants, it has been suggested by the staff of the U.S. EPA's Office of Air Quality Planning and Standards (1982) that an averaging time of 1 to 3 hours would be most appropriate for effects on vegetation since short-term peaks of NO_2 cause as much if not more damage to vegetation than does exposure over a growing season. As well, the OAQPS staff noted that by meeting a 1 to 3 hour standard at appropriate levels, there is a high probability of protecting against longer term effects based upon the relationship of short-term peaks to long-term means.

TABLE 4.3-4 PROJECTED NO_2 EXPOSURES THAT MAY INDUCE 5 PERCENT FOLIAR INJURY LEVELS ON SELECTED VEGETATION (HECK AND TINGEY, 1979)

Time (hr)	Concentrations Producing Injury					
	Susceptible[a]		Intermediate[a]		Tolerant[a]	
	ppm	mg/m^3	ppm	mg/m^3	ppm	mg/m^3
0.5	6-10	11.28-18.80	9-17	16.92-31.96	≥ 16	≥ 30.08
1.0	4-8	7.52-15.04	7-14	13.16-26.32	≥ 13	≥ 24.44
2.0	3-7	5.64-13.16	6-12	11.18-22.56	≥ 11	≥ 20.68
4.0	2-6	3.76-11.28	5-10	9.40-18.80	≥ 9	≥ 16.92
8.0	2-5	3.76- 9.40	4-9	7.52-16.92	≥ 8	≥ 15.04

[a] Plant type.

1 ppm = 1.88 mg/m^3

Source: EPA 1982b

Growth and yield experimental data on NO_2 effects are much less comprehensive than foliar injury information, but foliar injury is in effect the end result of internal effects and may be considered a rough measure of growth and yield parameters. NO_2- induced alterations in assimilatory capacity of plants through altered metabolism in leaf injury may also affect the growth of plants (OAQPS, 1982; Maclean, 1977).

Several individual studies on NO_2 effects on crop yield and growth have been summarized by U.S. EPA (1982b) and OAQPS (1982). Table 4.3-5 provides a summary of such effects, including certain foliar and physiological responses as well. Table 4.3-6 reports data on chronic NO_2 exposures on growth and yield from Zahn (1975).

As noted earlier, because of the inter-relationships of dose and time, there is no single threshold dose for an effect (U.S. EPA, 1982b). Maclean (1975) summarized the literature to illustrate interaction of NO_2 concentration, and exposure duration (dose) for metabolic and growth effects, as well as for foliar lesions and death. These threshold curves are illustrated in Figure 4.3-1, including both acute and chronic NO_2 doses. The death curve is drawn from a small database and is short. NO_2 doses approaching this threshold result in complete defoliation of some species, but are not lethal. The threshold curve for foliar injury is based on observations at many NO_2 doses, and the shift in leaf injury from necrosis to chlorosis for doses along this curve generally occurred between 10 and 100 hours. Because no measurable effects have been reported for NO_2 doses below the lower curve, the U.S. EPA (1982b) considered it as the threshold metabolic and growth effects. Doses in the area between this curve and that for leaf injury represent those that do not injure leaves but often result in growth suppression or metabolic effects.

Detection of injury from pollutants often requires the measurement of subtle responses in processes such as photosynthesis, transpiration and rates of metabolic activities (OAQPS, 1982). Examples include recent work by Law and Mansfield (1981), but the relationships

TABLE 4.3-5

TABULAR SUMMARY OF EFFECTS OF NITROGEN DIOXIDE ON VEGETATION

Exposure Duration (Continuous)	Concentration ppm	mg/m^3	Plant Species	Plant Response	Reference
90 min.	0.66	1.24	oat (*Avena sativa* var. Park) alfalfa (*Medicago sativa* var. Ranger)	Inhibition of rate of net photosynthesis	Hill and Bennett, 1970
20 hrs.	0.25	0.47	tomato (*Lycopersicon esculentum* var. Moneymaker)	Inhibition of rate of net photosynthesis	Capron and Mansfield, 1976
10-22 days	0.15-0.26	0.28-0.49	tomato (*Lycopersicon esculentum*)	Decrease in dry weight and leaf area; darker green color and downward curvature of leaves	Taylor and Eaton, 1966
35 days	0.5	0.94	navel oranges (*Citrus sinensis* Osbeck)	Severe defoliation and leaf chlorosis	Thompson et al., 1970
8.5 months	0.25	0.47	navel oranges (*Citrus sinensis* Osbeck)	Increased defoliation and reduced yield	Thompson et al., 1971
128 days	0.25	0.47	tomato (*Lycopersicon esculentum* var. Moneymaker)	Decrease in fresh weight yield (22%) average fruit wt (12%), and the number of fruit (11%)	Spierings, 1971
Exposed continuously for 5 days per week (103.5 hrs/wk for 20 wks)	0.11	0.21	Orchard grass (*Dactylis glomerata* var. Aberystwyth S37)	21% reduction in leaf area, 7% reduction in dry weight of green leaves	Ashenden, 1979, 1980
Exposed continuously for 5 days per week (103.5 hrs/wk for 20 wks)	0.11	0.21	Kentucky bluegrass (*Poa pratensis* var. Monopoly)	17% reduction in leaf area, 29% reduction in dry weight of green leaves	Ashenden, 1979, 1980
Exposed continuously for 5 days per week (103.5 hrs/wk for 20 wks)	0.11	0.21	Italian ryegrass (*Lolium multiflorum* var. milamo)	1% increase above the control in leaf area, 10% reduction in dry weight of green leaves	Ashenden, 1979, 1980
Exposed continuously for 5 days per week (103.5 hrs/wk for 20 wks)	0.11	0.21	Timothy (*Phleum pratense* var. Eskimo)	30% increase above control in reduction in leaf area, 14% increase above control in reduction in dry weight of green leaves	Ashenden, 1979, 1980

Source: OAQPS 1982

TABLE 4.3-6

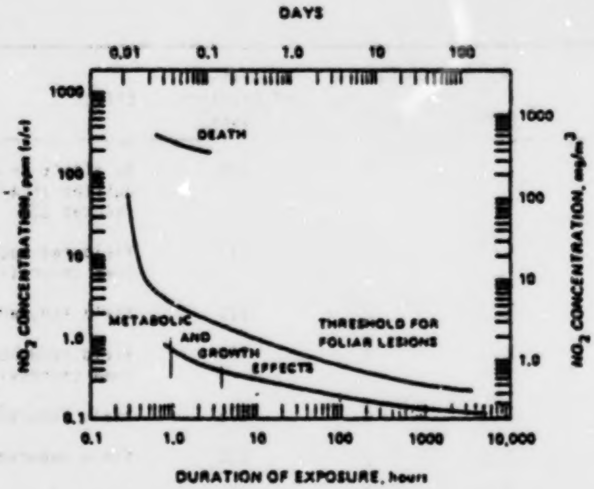
EFFECTS OF CHRONIC NO_2 EXPOSURES ON PLANT GROWTH AND YIELD (ZAHN, 1975)

Plant Type	NO_2 Concentration ppm	NO_2 Concentration mg/m^3	Duration of Exposure (hours)	Effect
Wheat	1.1	2	334	No effect on grain yield, but the straw yield was reduced 12%.
Bush Bean	1.1	2	639	Yield reduced 27%; Some chlorosis
Endive	1.1	2	620	Yield reduced 37%
Carrot	2.1	4	357	Yield reduced 30%; Some chlorosis
Radish	2.1	4	278	Yield reduced 13%
Currant	1.1	2	213	Yield reduced 12%
Roses	2.1	4	357	No injury
European Larch	1.1	2	537	No injury
Spruce	1.1-1.6	2-3	1900	7% decrease in linear growth. Growth was decreased 17% in the year following the exposure.

[a] Necrosis did not occur on any plants.

Source: EPA 1982b

FIGURE 4.3-1



Threshold curves for the death of plants, foliar lesions, and metabolic or growth effects as related to the nitrogen dioxide concentration and the duration of exposure (McLean, 1975).

Source: Maclean, 1975 as cited in U.S. EPA 1982 b between these physiological effects and more obvious changes in growth or leaf condition have not yet been established in a quantitative manner. Physiological changes such as reduction in photosynthetic rate occur in some species after being exposed to low levels of NO_2 before there is any visible injury (OAQPS, 1982).

Burton et al. (1981) have summarized data relating to the effects of nitrogen dioxide on vegetation to assist in the re-evaluation of the Canadian air quality objectives for that substance. Data lists for effects and no effects are provided in Tables 4.3-7 and 4.3-8 respectively, while the data are presented graphically in Figures 4.3-2 and 4.3-3. Table 4.3-9 provides those authors' summary of effects of NO_2 on vegetation based upon a literature review.

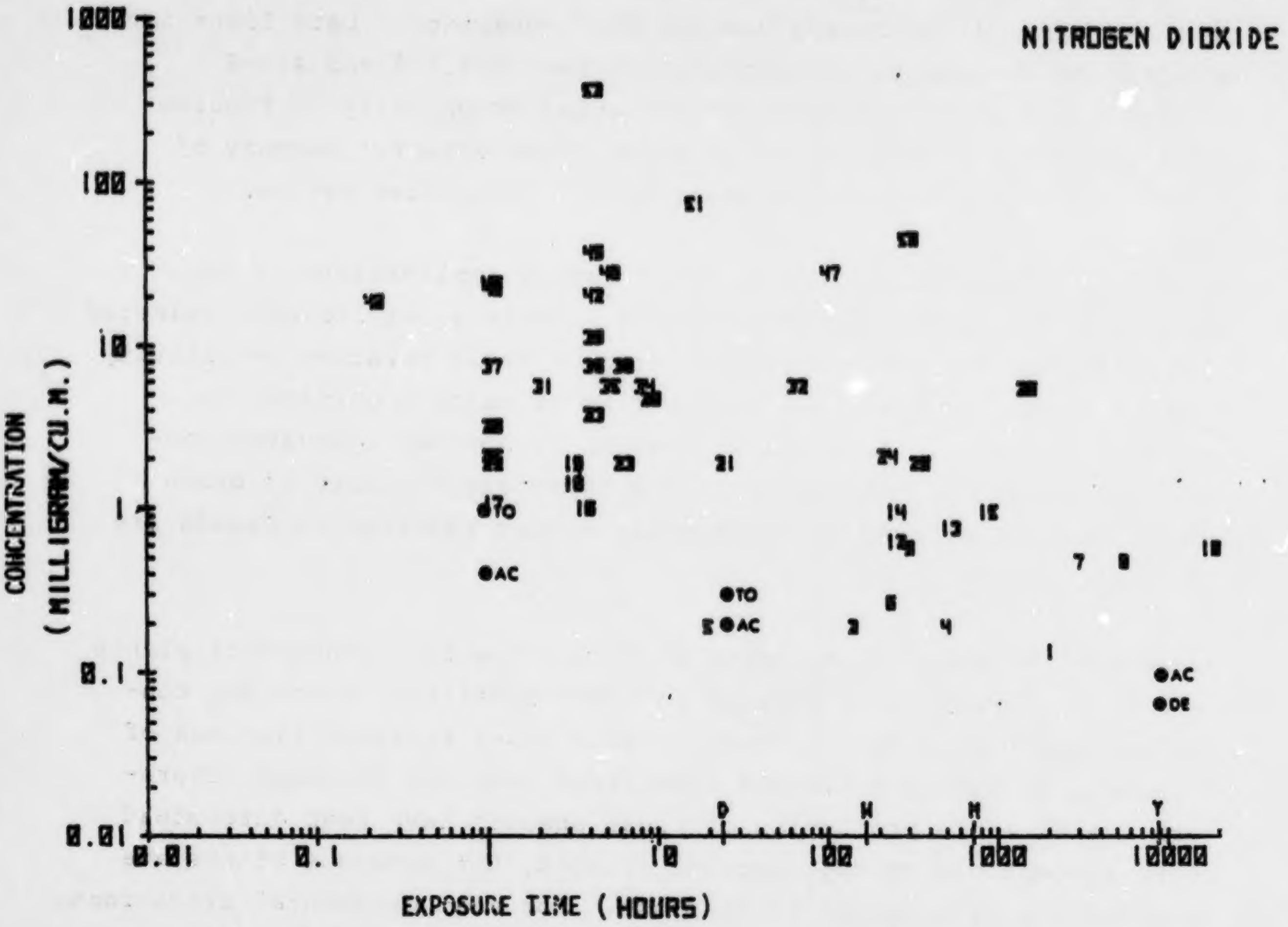
Forestry: Specific studies on the forestry implications of NO_2 effects do not appear to be available. Table 4.3-1 includes selected trees, shrubs and fruit trees in terms of their relative sensitivity. Most of these, however, are not species of major importance to Canadian forestry. As noted in Chapter 2, however, elevated concentrations of NO_x only occur in the immediate vicinity of urban areas, so that effects on harvestable timber reserves in Canada are unlikely.

Ornamental Plants: In contrast to forest species, ornamental plants tend to be largely concentrated in urban localities where NO_x concentrations are relatively high. Table 4.3-1 includes listings of ornamental shrubs and flowers summarized from the research literature. A sizeable proportion of these species have been determined to be susceptible to NO_2 exposure effects, but numerous others are intermediate or tolerant in response. For most ornamental situations, substitutions could be made from among the more tolerant species if NO_2 effects were thought to be resulting in foliar injury and aesthetic degradation.

From Table 4.3-4, 5 percent foliar injury would be anticipated in susceptible species from doses ranging from 0.5 hours at 11.3 to 18.8 mg/m^3 (6 to 10 ppm) NO_2 to 8 hours at 3.8 to 9.4 mg/m^3 (2 to 5 ppm) NO_2. If an

FIGURE 4.3-2

CRITERIA PLOT OF EFFECTS OF NITROGEN DIOXIDE ON ALL TYPES OF VEGETATION



KEY

Existing Canadian NO_2 Objectives

DE = desirable, AC = acceptable
TO = tolerable

Numbers on plot represent numbers of experiments reviewed showing effects at that concentration and duration of exposure

See Table 4.3-7 for data

Source: Burton et al. (1981)

TABLE 4.3-7

Data List for Effects of Nitrogen Dioxide on All Types of Vegetation

POLLUTANT : NITROGEN DIOXIDE

CONCENTRATION MG/M13	CONCENTRATION PPM	TIME HOURS	EXPOSURE TYPE	#	REFERENCE	CATEGORY
● 0.13	0.07	2070.00	1	1	ASHENDEN & MANSFIELD 1978	VN0
● 0.19	0.10	144.00	C	2	HORSMAN & WELLBURN 1975	VE0
● 0.19	0.10	144.00	C	3	WELLBURN ET AL 1976	VE0
● 0.19	0.10	504.00	C	4	WELLBURN ET AL 1976	VE0
● 0.19	0.10	20.00	C	5	CAPRON & MANSFIELD 1976	VE0
0.26	0.14	240.00	C	6	TAYLOR & EATON 1966	VE0
0.47	0.25	3120.00	C	7	SPIERINGS 1971	VE0
0.47	0.25	5760.00	C	8	THOMPSON ET AL 1970	VTU
0.56	0.30	312.00	C	9	FUJIWARA & ISHIKAWA 1974	VE0
0.56	0.30	17520.00	C	10	HELMS ET AL 1970	VTU
0.56	0.30	17520.00	C	11	HELMS ET AL 1970	VO0
0.62	0.33	240.00	C	12	TAYLOR & EATON 1966	VE0
0.75	0.40	504.00	C	13	SPIERINGS 1971	VE0
0.94	0.50	240.00	C	14	SPIERINGS 1971	VE0
0.94	0.50	840.00	C	15	THOMPSON ET AL 1970	VT0
1.00	0.53	3.50	1	16	ZAHN 1975	VF
● 1.07	0.57	1.00	C	17	MASARU ET AL 1976	VO0
1.41	0.75	3.00	C	18	FAIRFAX & LEPP 1976	VN0
1.88	1.00	3.00	C	19	TROIANO & LEONE 1974	VE0
1.88	1.00	336.00	C	20	TAYLOR ET AL 1975	VE0
1.98	1.00	24.00	C	21	WELLBURN ET AL 1972	VE0
1.88	1.00	1.00	C	22	WELLBURN ET AL 1972	VE0
1.88	1.00	6.00	C	23	NASH 1976	VN0
2.07	1.10	216.00	C	24	ZAHN 1975	VE0
2.07	1.10	1.00	C	25	ZAHN 1975	VE0
3.20	1.70	1.00	C	26	MASARU ET AL 1976	VO0
3.76	2.00	4.00	C	27	DUNNING ET AL 1970	VE0
4.70	2.50	8.70	C	28	TAYLOR & EATON 1966	VE0
5.45	2.90	1440.00	C	29	HELMS ET AL 1970	VO0
5.45	2.90	1440.00	C	30	HELMS ET AL 1970	VE0
5.64	3.00	2.00	C	31	HILL & BENNETT 1970	VE0
5.64	3.00	64.00	1	32	MATSUSHIMA ET AL 1977	VT0
3.76	2.00	4.00	C	33	TINGEY ET AL 1971	VE0
5.64	3.00	8.00	C	34	MIDDLETON ET AL 1958	VE0
5.64	3.00	5.00	C	35	SRIVASTAVA ET AL 1974,1975	VE0
7.52	4.00	4.00	C	36	FUJIWARA & ISHIKAWA 1974	VE0
7.52	4.00	1.00	C	37	ZEEVART 1974	VE0
7.52	4.00	6.00	C	38	NASH 1976	VN0
11.28	6.00	4.00	C	39	TAYLOR ET AL 1975	VE0
18.80	10.00	0.20	C	40	MACLEAN ET AL 1968	VO0
18.80	10.00	0.20	C	41	MACLEAN ET AL 1968	VT0
20.49	10.90	4.00	C	42	TAYLOR & EATON 1966	VE0
22.56	12.00	1.00	I	43	MATSUSHIMA 1977	VT0
22.56	12.00	1.00	I	44	MATSUSHIMA 1977	VE0
24.44	13.00	1.00	C	45	MATSUSHIMA 1973	VT0
24.44	13.00	1.00	C	46	MATSUSHIMA 1973	VO0
28.20	15.00	100.00	C	47	BROOKS & CSALLANY 1978	VE0
28.20	15.00	5.00	C	48	FUJIWARA & UMEZAWA 1975	VE0
37.60	20.00	4.00	C	49	FUJIWARA & ISHIKAWA 1974	VE0
45.12	24.00	288.00	C	50	CHAKRABARTI 1976	VE0
75.20	40.00	16.00	C	51	MATSUSHIMA 1972	VT0
376.00	200.00	4.00	C	52	MACLEAN ET AL 1968	VO0
376.00	200.00	4.00	C	53	MACLEAN ET AL 1968	VT0
18800.00	10000.00	1.00	C	54	LOPATA & ULLRICH 1975	VE0

●Paper felt by Burton *et al*.(1981)
to be critical to revision of existing
objectives.

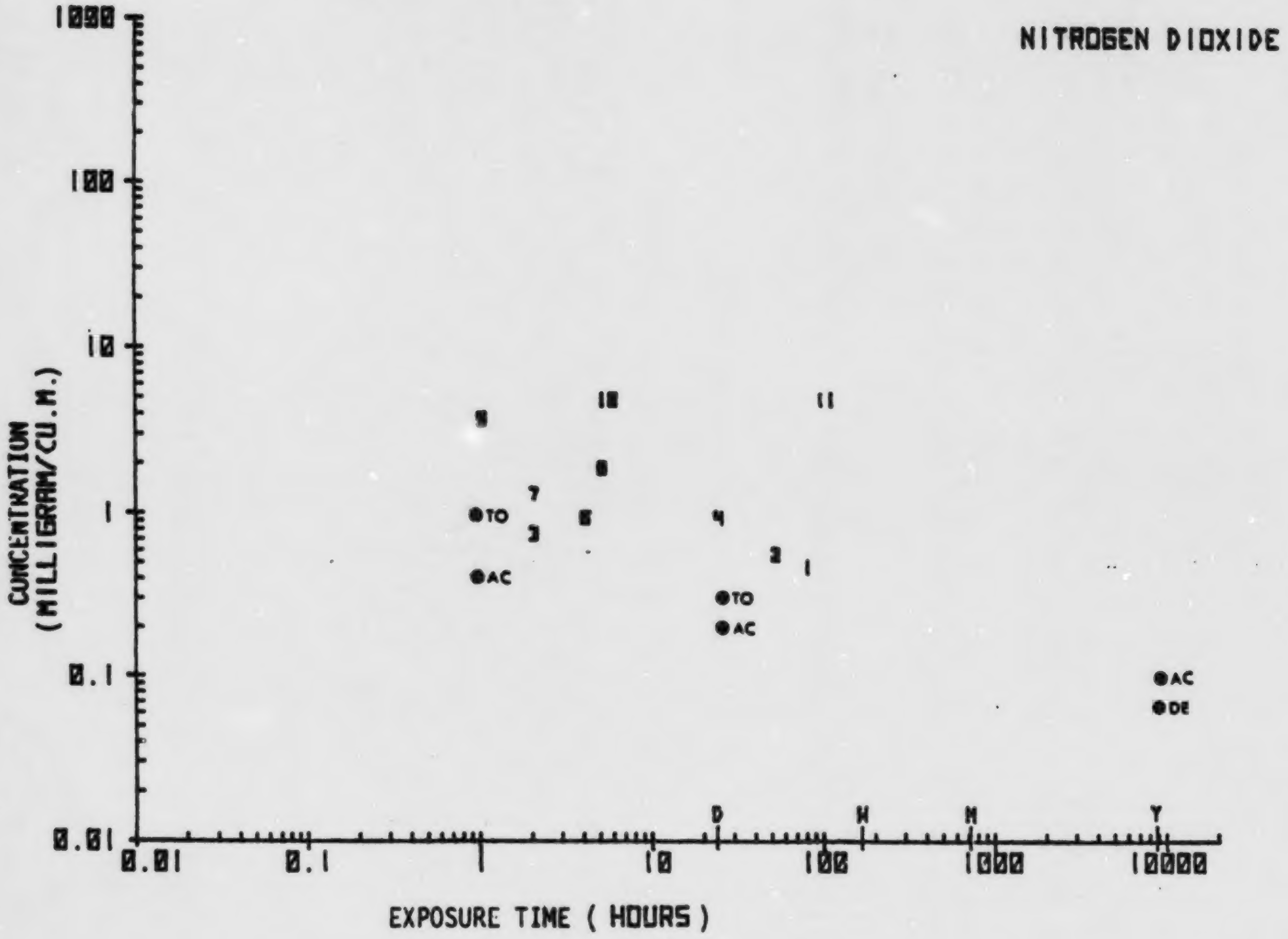
Exposure Type: C - continuous
I - intermittent

Category: V = vegetation, E = economic
N = natural, O = ornamental,
T = trees, 0 = effect
11 = no effect

Source: Burton *et al*. (1981)

FIGURE 4.3-3

CRITERIA PLOT OF NO EFFECTS OF NITROGEN DIOXIDE ON ALL TYPES OF VEGETATION



Kev - sec Figure 4.3-2

See Table 4.3-8 for data

Source: Burton et al. (1981)

TABLE 4.3-8

Data List for No Effects of Nitrogen Dioxide on All Types of Vegetation

POLLUTANT : NITROGEN DIOXIDE

CONCENTRATION MG M↑3	PPM	TIME HOURS	EXPOSURE TYPE	#	REFERENCE	CATEGO
● 0.47	0.25	80.00	C	1	TROIANO & LEONE 1977	VE
● 0.56	0.30	52.00	C	2	TROIANO & LEONE 1974	VE
● 0.75	0.40	2.00	C	3	HILL & BENNETT 1970	VE
● 0.94	0.50	24.00	C	4	YAMAZOE & MAYUMI 1977	VE
● 0.94	0.50	4.00	C	5	TINGEY ET AL 1977	[illegible]
● 0.94	0.50	4.00	C	6	TINGEY ET AL 1977	N
● 1.32	0.70	2.00	C	7	HILL & BENNETT 1970	VE
● 1.88	1.00	5.00	C	8	FUJIWARA & UMEZAWA 1975	E
● 3.76	2.00	1.00	C	9	BENNETT ET AL 1975	[illegible]
● 4.93	2.62	5.00	C	10	TAYLOR & EATON 1966	VE
● 4.93	2.62	96.00	C	11	TAYLOR & EATON 1966	E

Key - See table 4.3-7

Source: Burton et al. (1981)

TABLE 4.3-9

SUMMARY OF EFFECTS OF NITROGEN DIOXIDE ON VEGETATION

NO_2 PPM	NO_2 MG/M3	LENGTH OF EXPOSURE	SPECIES	EFFECTS	COMMENTS	SAMPLE NUMBERS EXPTAL	SAMPLE NUMBERS CONTROL	REFERENCE
0.0625, 0.125, 0.25	0.1175, 0.235, 0.47	9.5 mos	Navel orange (*Citrus sinensis*)	Appeared to be a trend towards increased leaf and fruit drop and decreased yield but was not significant statistically. When 3 data sets analyzed together, leaf and fruit drop and yield were significantly changed.	Analysis of 3 data sets together statistically questionable. See 0.5 ppm exposure.	19	12	Thompson et al. 1970
0.11	0.21	103.5 h/w, 20 w	Cocksfoot (*Dactylis glomerata*) Italian ryegrass (*Lolium multiflorum*) Timothy grass (*Phleum pratense*) Meadow grass (*Poa pratensis*)	Yield (total dry weight) of cocksfoot and meadow grass reduced by NO_2, other species not significantly affected. No significant reductions in leaf area.		10 plants/ treatment		Ashenden and Mansfield 1978
0.1,0.25, 0.5	0.19,0.47 0.94	20 h	Tomato (*Lycopersicon aesculentum*)	Mean rate of photosynthesis (mg CO_2 $dm^{-2}h^{-1}$) at 0.1 ppm was not statistically significant. Rate of photosynthesis was significantly reduced at higher concentrations.				Capron and Mansfield 1976
0.1,1	0.19,1.88	6 d	Pea (*Pisum sativum*)	Chlorophyll content increased significantly at 1 ppm level only. No significant differences in the activity of ribulose diphosphate 1,5-carboxylase (RuDPC) glutamate-pyruvate transaminase (GPT), glutamate-oxaloacetate transaminase, (GOT) or peroxidase.	Plants were 9 days old.	20	20	Horsman and Wellburn 1975
0.1,0.2, 1	0.19, 0.38, 1.88	6 d	Pea (*Pisum sativum*)	The only significant changes found were: decrease in glutamate oxaloacetate transaminase (GOT) at 0.1 ppm, an increase in glutamate dehydrogenase (GDH) at 0.2 ppm, increase in ribulose-1,5-diphosphate carboxylase (RuDPC) at 1 ppm	Plants were 2 ws. old.			Wellburn et al. 1976
0.1,0.5	0.19,0.94	3 w	Tomato (*Lycopersicon aesculentum*)	Stimulated glutamate pyruvate transaminase (GPT) activity at low concentration, no effect at high concentration. Inhibited glutamate oxaloacetate transaminase (GOT), peroxidase activity at both concentrations. RuDPC activity decreased at 0.1 ppm, increased at 0.5 ppm. No effect on nitrite reductase.				Wellburn et al. 1976

cont'd

TABLE 4.3-9 (CONT'D)

NO_2 PPM	NO_2 MG/M3	LENGTH OF EXPOSURE	SPECIES	EFFECTS	COMMENTS	SAMPLE NUMBERS EXPTAL	SAMPLE NUMBERS CONTROL	REFERENCE
a. 0.14-0.28 b. 0.16-0.23 c. 0.28-0.62 d. 0.44-0.55	a. 0.27-0.53 b. 0.31-0.43 c. 0.53-1.17 d. 0.82-1.03	a. 22 d b. 10 d c. 14 d d. 19 d	Tomato (*Lycopersicon aesculentum*)	Decrease in dry weight of plant material and leaf area. Increase in chlorophyll content, downward curvature of leaves. Nitrate nitrogen levels decreased.	Concentration varied during each experiment. See 0.33, 2.5, 2.62, 10.9 ppm exposures.	20	20	Taylor and Eaton 1966
0.15	0.28	30 or 60 min	Lily (*Lilium longiflorum*)	9.6% and 20% (respectively) inhibition in pollen tube elongation.	See 0.57, 1.7, 2 ppm exposures.			Masaru et al. 1976.
0.25	0.47	130 d	Tomato (*Lycopersicon aesculentum*)	Decrease in fresh weight yield (22%); 12% reduction in fruit weight, 11% in fruit number. Smaller leaves and petioles, yellowing of older leaves, early leaf drop.	See 0.4, 0.5 ppm exposures.	26	26	Spieringa 1971
a. 0.25 b. 0.40	a. 0.47 b. 0.76	a. 80 h b. 164 h	Tomato (*Lycopersicon aesculentum*)	No NO_2 injury was found on the fumigated plants, irrespective of the NO_3-N content of the nutrient medium. Total N had increased following exposure especially in the leaves of plants subjected to the higher N-NO_3 content of the medium.				Troiano and Leone 1977
0.3, 0.6	0.56, 1.13	13-55 d	11 types of plants.	Leaf fall in eggplants. Small leaves in buckwheat. Inhibited growth of eggplant, buckwheat and turnips. Rice plants increased in growth and yield.	See 4, 20 ppm exposures.			Fujiwara and Ishikawa 1974
up to 0.3	up to 0.56	several years	Indigenous vegetation in Chattanooga	NO_2 damage on leaves of honeysuckle, rosebush, tulips, euonymous and pine in 1967. Pine, euonymous, dogwood and honeysuckle showed damage in 1968.	SO_2, ozone, PAN, HF and other pollutants also present in ambient air. See 2.9 ppm exposure.			Helms et al. 1970
0.3	0.56	52 h	Tobacco (*Nicotiana glutinosa*)	No NO_2 injury (presumably visual).	See 1 ppm exposure.			Troiano and Leone 1974 Abstract.
0.33-0.44, up to 0.55 at night	0.62-0.82, up to 1.03 at night	10-19 d	Pinto bean (*Phaseolus vulgaris*)	Decrease in fresh and dry weights. Increase in chlorophyll content per unit weight, (but not per leaf). Downward cupping of leaves and darker green colouration.	NO_2 absorption may be less at night. Concentration varied during experiment. See 0.14, 2.5, 2.62, 10.9 ppm exposures.			Taylor and Eaton 1966
0.4	0.75	2 h	Oats (*Avena sativa*)	No reduction in net assimilation.	See 0.7, 3 ppm exposures.			Hill and Bennett 1970
0.4-0.5	0.75-0.94	21-45 d	Tomato (*Lycopersicon aesculentum*)	Taller plants, smaller leaves and petioles.	See 0.25 ppm exposure.			Spieringa 1971

(cont'd

TABLE 4.3-9 (CONT'D)

NO_2 PPM	MG/M3	LENGTH OF EXPOSURE	SPECIES	EFFECTS	COMMENTS	SAMPLE NUMBERS EXPTAL	CONTROL	REFERENCE
0.5	0.94	10 d	Tomato (Lycopersicon aesculentum)	Taller plants, thinner stems, smaller leaves and reduced fresh weights of entire plants.				Spierings 1971
0.5,1	0.94,1.9	35 d	Navel orange (Citrus sinensis)	Severe defoliation occurred along with chlorosis of remaining tree leaves.	See 0.0625 ppm exposure.	19	12	Thompson et al. 1970
0.5-4	0.94-7.5	4 h	Western wheat grass (Agropyron smithii)	1% injury at 2 and 4 ppm.	Foliar injury assessed 96 h after exposure.	9		Tingey et al. 1977
			Blue gramma (Bonteloua gracilis)	3% injury at 1 ppm, 4% at 2 ppm, 8% at 4 ppm.		9		
			Prairie June grass (Koeleria cristata)	1% injury at 2 ppm, 4% at 4 ppm.		9		
			Needle and thread grass (Stipa comata)	1% injury at 2 ppm, 1% at 4 ppm.		9		
			Fringed sagewort (Artemesia frigida)	No injury.		9		
			Wheat (Triticum aestivum)	1% injury at 4 ppm.		9		
0.5	0.94	24 h sativa)	Paddy (Oryza stem damage.	No effects in terms of leaf injury or				Tamazoe and Mayumi 1977
			Corn (Zea mays)	No effect in terms of leaf injury.				
0.57	1.07	1, 2 and 5 h	Lily (Lilium longiflorum)	11, 14 and 20% (respectively) inhibition in pollen tube elongation.	See 0.15, 1.7, 2 ppm exposures.			Masaru et al. 1976
0.7	1.3	2 h	Alfalfa (Medicago sativa)	No reduction in net assimilation.	See 0.4, 3 ppm exposures.			Hill and Bennett 1970
0.21-0.85	0.4-1.6	3-6 h	Moss	Effects on exchangeable cation levels. Reduced magnesium loss.				Fairfax and Lepp 1976
0.8, 1.6 2.39, 3.19	1.5, 3.0 4.5, 6.0	daylight hours for 3 w	Sunflower (Helianthus annuus)	Increases seen in plant height and in dry weight of leaves, stems and roots.	NO_2 was the only nitrogen source.			Faller 1972

(cont'd)

TABLE 4.3-9 (CONT'D)

NO_2		LENGTH OF EXPOSURE	SPECIES	EFFECTS	COMMENTS	SAMPLE NUMBERS		REFERENCE
PPM	MG/M3					EXPTAL	CONTROL	
1,10	1.9, 19	5 h	Spinach a) Minsterland variety b) Hoyo variety	No symptoms of damage.	See 15 ppm exposure.			Fujiwara and Umezawa 1975
1,2	1.9,3.8	6 h	*Anaptychia neoleucomelaena* *Lecanora chrysoleuca* *Parmelia praesignis* *Usnea cavernosa*	No effect on chlorophyll concentration in these lichens.	Chlorophyll extractions were made 12 h. after fumigation. See 4 ppm exposure.			Nash 1976
1	1.9	14 d	Bush beans	Significant growth depression.	See 6 ppm exposure.			Taylor et al. 1975
1-1.5	1.9-2.8	3 h	Tobacco (*Nicotiana glutinosa*)	Plants grown in medium with high N soil were severely damaged whereas plants grown in low N soil were not damaged.	See 0.3 ppm exposure.			Troiano and Leone 1974 Abstract.
1	1.9	1 d	Beans (*Vicia faba*)	Swelling of thylakoids in chloroplasts.				Wellburn et al. 1972
1-3	1.9-5.6	1 h	Beans (*Vicia faba*)	Swelling of thylakoids in chloroplasts - apparently reversible.				Wellburn et al. 1972
1.1-2.1	2-4	213-1300 h	10 garden plants	Some loss of productivity due to reduced leaf size and slight yellowing in some plants. Yield reductions up to 37%. e.g. 12% reduction in yield of currants exposed for 213 h to 1.1 ppm NO_2.				Zahn 1975
1.7	3.2	1, 2 and 5 h	Lily (*Lilium longiflorum*)	22, 40 and 80% (respectively) inhibition in pollen tube elongation.	See 0.15, 0.57, 2 ppm exposures.			Masaru et al. 1976
2-10	3.8-19	1-3 h	Oat (*Avena sativa*) Swiss chard (*Beta vulgaris*) Sweet pea (*Raphanus sativa*) Pea (*Pisum sativum*)	No visible injury.	Dicotyledons: 30 plants/treatment. Monocotyledons: 60-100 plants/treatment.			Bennett et al. 1975
2	3.8	4 h	6 crop species tomato, radish, oats, tobacco, pinto bean, soybean.	Threshold for injury; marginal and interveinal bifacial necrosis.				Dunning et al. 1970 Abstract.

(cont'd

TABLE 4.3-9 (CONT'D)

NO_2 PPM	MG/M3	LENGTH OF EXPOSURE	SPECIES	EFFECTS	COMMENTS	SAMPLE NUMBERS EXPTAL	CONTROL	REFERENCE
2	3.8	1, 2 and 5 h	Lily (Lilium longiflorum)	62, 100 and 100% (respectively) inhibition in pollen tube elongation.	See 0.15,0.57, 1.77 ppm exposures.			Masaru et al. 1976
2	3.76	4 h	Tobacco, bean, tomato, radish, oat, soybean	Bifacial, marginal and/or interveinal necrosis to broad-leaf plants. Oats developed tipburn and necrotic streaking of the leaf blade.				Tingey et al. 1971
2.5	4.72	8.7 h	Nicotiana glutinosa	Collapsed, bleached, dead tissue at apex and leaf margins. Most severe on older leaves.	See 0.14, 0.33, 10.9 ppm exposures.			Taylor and Eaton 1966
2.62	4.93	4 d	3 strains of tobacco	No visible damage.				Taylor and Eaton 1966
up to 2.9	up to 5.5	60 d	Gladioli, tobacco, petunia, geranium, begonia, pinto beans, tomato	Tomato damage occurred as dark green colour and downward leaf curl. Tobacco also damaged probably by a mixture of pollutants. Growth suppression observed.	SO_2, ozone, PAN, HF and other pollutants also present in ambient air. See 0.3 ppm exposure.			Helms et al. 1970
3, 5.4	5.6,10.2	2 h	Oats (Avena sativa) Alfalfa (Medicago) sativa)	25% and 50% reduction, respectively, in net assimilation. Recovery from 5.4 ppm required more than 4 h.	See 0.4, 0.7 ppm exposures.			Hill and Bennett 1970
3	5.64	8 h/d, 8 d	Zelkova trees	Leaves appeared normal. All membranes normal. Chloroplast lamellae seemed to be dissolving.				Matsushima et al. 1977
3	5.6	5 h	Bean (Phaseolus vulgaris)	Leaf photosynthesis studied at CO_2 concentrations between 100 and 600 ppm. At 100 ppm CO_2, photosynthesis inhibited 55%; at 500 and 600 ppm CO_2 15-17% inhibition of photosynthesis. Rate of transpiration little affected by NO_2; NO_2 inhibited rate of dark respiration and photorespiration.				Srivastava et al. 1975
4-10	7.52-18.8	4-8 h	17 types of plant	In soybeans and buckwheat: waxy leaf surface, interveinal grayish-or yellowish-white areas. In grapes, black-brown spots day after exposure.	See 0.3, 20 ppm exposures.			Fujiwara and Ishikawa 1974

(cont'd)

TABLE 4.3-9 (CONT'D)

NO₂		LENGTH OF EXPOSURE	SPECIES	EFFECTS	COMMENTS	SAMPLE NUMBERS		REFERENCE
PPM	MG/M3					EXPTAL	CONTROL	
4,8	7.52, 15	6 h	Anaptychia neoleucomelaena, Lecanora chrysoleuca, Parmelia praesignis, Usnea cavernosa	Significant reductions in chlorophyll in these lichens.	Chlorophyll extractions were made 12 h. after fumigation. See 1 ppm exposure.			Nash 1976
4,12	7.5, 23	60 min	Pea (Pisum sativum) Bean (Phaseolus vulgaris)	NO_2 induced a considerable nitrate reductase activity for both 4 and 12 ppm, even after 10 minutes.	Plants were 4 w old.			Zeevaart 1974
6	11.3	4-8 h	Peas, alfalfa, bush beans	Leaf lesions. Moderately severe necrosis.	See 1 ppm exposure.			Taylor et al. 1975
10,100, 1000	18.8, 188, 1880	1 h	Oat (Avena sativa) potato (Solanum tuberosum), Sugar beet (Beta sp.)	Toxic dose for sugar beets was 100 ppm during daylight fumigations and 10 ppm at night. After 1000 ppm exposure, fresh weight of sugar beet roots was 1/3 less than that of the of the controls. 100 ppm exposure caused abscission of potato flowers and lodging of oats.				Czech and Nothdurft 1952
10-250	19-470	8-0.2 h	Ornamentals and Citrus species	Exposures tabulated in ppm-hours (ppm x hours). No visible symptoms evident on plants exposed to lowest treatment, 10 ppm-hours (40 ppm for 0.25 hour). Highest exposure, 375 ppm-hours (150 ppm for 2.5 hours) resulted in necrosis on 90-100% of leaf surface area.				MacLean et al. 1968
10.9	20.53	4 h	Pinto bean (Phaseolus vulgaris)	Visible symptoms above this level.	See 0.14, 0.3, 2.5, 2.62 ppm exposures.			Taylor and Eaton 1966
12	23	3 h/d until visual symptoms appeared.	Fruit trees, ornamental trees, vegetables, herbaceous plants.	Some plants showed injury after a 1 hr exposure while some showed no symptoms after 21 h				Matsushima 1977
15	28	100 h	Corn seeds Soybean seeds	Tocopherol destruction; no formation of polyunsaturated fatty acids (PUFA) or lipofuscin.				Brooks and Csallany 1978

(cont'd)

TABLE 4.3-9 (CONT'D)

NO_2 PPM	MG/M3	LENGTH OF EXPOSURE	SPECIES	EFFECTS	COMMENTS	SAMPLE NUMBERS EXPTAL CONTROL	REFERENCE
15, 20	28.2, 37.6	5 h	Spinach a) Minsterland variety b) Hoyo variety	Symptoms of damage appeared on Minsterland variety at 20 ppm and on Hoyo variety at 15 ppm.	See 1 ppm exposure.		Fujiwara and Umezawa 1975
20-50	37.6-94	4 h	10 weed species	Most sensitive species injured at 20 ppm, least sensitive at 50 ppm. Symptoms were irregular white to brown collapsed tissue near the margins. Plants grown in moist soil showed large injury while little injury developed under conditions of moisture stress.			Benedict and Breen 1955
20	37.6	4-8 h	17 types of plant	Damage to almost all plants.	See 0.3, 4 ppm exposures.		Fujiwara and Ishikawa 1974
24	45	12 d	Garden pea (Pisum sativum) String bean (Phaseolus vulgaris)	Bean: rate of germination delayed 48 h, per cent germination also decreased. No effects on pea germination.			Chakrabarti 1976
40	75.2	16 h	Citrus natsudaidai	Slight visible injury. Amino acid assimilation stimulated, especially alanine and glutamic acid. Organic acid decreased.			Matsushima 1972
10,000	18,800	30 min - 15 h	String bean (Phaseolus vulgaris)	2 h exposure-chloroplasts showed invaginations; assimilation rate decreased to about 50% of the control. 15 h exposure - total breakdown of the fine structure of the cells.			Dolzmann and Ullrich 1966
10,000	18,800	1 or 2 h	bush bean	Impaired photosynthesis at 2 h only. Mitochondria trapped in invaginations of chloroplasts. Dense layers of filaments in stroma. Pigment changes observed.			Lopata and Ullrich 1975

Source: Burton et al. (1981)

average exposure duration of 2 hours were considered to be appropriate, then susceptible species would be damaged by 5.6 to 13.2 mg/m^3 (3 to 7 ppm), while tolerant species would be able to withstand over 20.1 mg/m^3 (11 ppm) NO_2 without sustaining 5 percent foliar injury.

Factors Affecting Responses to NO_x

Evaluations by OAQPS (1982) have determined that the most notable feature of the response of vegetation to NO_2 stress is the varied degrees of injury. These differing responses appear to be related to physiological processes affecting NO_2 uptake into the leaf, pollutant toxicity at target sites, and cellular repair capacity. The authors also noted that environmental conditions, as well as the condition or status of the plant itself, influence the response to NO_2. Findings have indicated that NO_2 susceptibility varies among species and even among varieties, cultivars, or clones of the same species, due to genetic factors.

Other studies have determined that another important biological factor affecting the severity of damage is the stage of development or age of the plant or plant part (OAQPS, 1982; U.S. EPA, 1982b). The main environmental factors affecting plant sensitivity to NO_2 include the presence or absence of other pollutants, soil moisture status, temperature, humidity, light intensity and time of day when exposure occurs.

One of the major factors affecting plant responses to NO_2 is the presence of sulphur dioxide (SO_2) and, to a lesser extent, other pollutants such as O_3 and PAN (Mansfield and Freer-Smith, 1981). A somewhat confusing picture emerged from the early investigations into the combined effects of NO_2 and SO_2. There were clear signs of severe damage caused by simultaneous exposures, but indications of the critical concentrations at which this might occur were far from clear, as were the dose-response relationships. Light regimes and plant nutrition, in addition to experimental techniques, appear to be the factor introducing variability in reported results.

Mansfield and Freer-Smith (1981) also noted that experimental fumigations with SO_2 and NO_2 in combination have shown that acute visible injury can be caused by concentrations that are well below the threshold for injury by either gas alone. A possible explanation may relate to the reduction of NO_2^- in leaves being inhibited by concentrations of SO_2 that normally accompany NO_2 pollution. NO_2^- is highly toxic and its accumulation in cells when SO_2 is present may be a major cause of injury.

Table 4.3-10 reports plant responses to NO_2 and SO_2 mixtures as summarized by OAQPS (1982). These data suggest that the injury threshold for NO_2 can be significantly decreased with the addition of SO_2, and these concentrations where observable injury has occurred were well within the ambient levels of NO_2 and SO_2 in some areas of North America. In addition to increased sensitivity to foliar injury from this mixture, experimental results have found synergistic growth and yield suppression (Ashenden, 1978, 1979, 1980) and physiological responses (Bull and Mansfield, 1974; White et al., 1974). OAQPS (1982) concluded that these are insufficient data on the combined effects of NO_2 and SO_2 to do a quantitative evaluation of yield reduction for various ambient exposure levels.

Quantitative Assessment of Effects

In the judgement of workers with the Strategies and Air Standards Division of the United States Environmental Protection Agency (OAQPS, 1982), there is insufficient evidence to demonstrate that exposure to NO_2 alone at low levels will lead to significant impacts on growth and yield for commercially important crops and indigenous vegetation.

Workers in the United Kingdom (Mansfield and Freer-Smith, 1981) and the United States (OAQPS, 1982) have concluded that while the combined effects of NO_2 and SO_2 may account for many observed air pollution effects on urban vegetation, there are insufficient data under all conditions to permit any quantitative evaluation of injury or reduction of yield.

TABLE 4.3-10

PLANT RESPONSE TO NITROGEN DIOXIDE AND SULFUR DIOXIDE MIXTURES

Exposure Duration	Concentration (ppm) NO_2/SO_2	Plant Response	Plant Species	Exposure Chamber[a]	Mixture Response[b]	References
1 hr.	0.5/0.5	0-5% foliar injury	radish (*Raphanus sativus* cv. Scarlet Globe)	CE	+	Bennett et al., 1975
1 hr.	0.05/0.05	Significantly decreased net photosynthesis	pea (*Pisum sativum*)	CE	0	Bull and Mansfield, 1974
2 hrs.	0.15/0.15	7% reduction in apparent photosynthesis. Some tissue death	alfalfa (*Medicago sativa* var. Ranger)	GH	+	White et al., 1974
2 hrs.	0.25/0.25	9% reduction in apparent photosynthesis	alfalfa (*Medicago sativa* var. Ranger)	GH	+	White et al., 1974
4 hrs.	0.10/0.10	0-10% foliar injury	tobacco (*Nicotiana tabacum* cv. Bel W_3)	GH	+	Heck, 1963
4 hrs.	0.05/0.05	0-2% foliar injury in 6 species	pinto (*Phaseolus vulgaris* cv. Pinto) oats (*Avena sativa* cv. Clintland 64) radish (*Raphanus sativus* cv. Cherry Belle) soybean (*Glycine max*, cv. Hark) tobacco (*Nicotiana tabacum* cv. Bel W_3) tomato (*Lycopersicon esculentum* cv. Roma VF)	GH	+	Tingey et al., 1971
same	0.10/0.05	9% foliar injury in tobacco. 0-1% foliar injury in 5 species.	same	GH	+	same
same	0.25/0.05	16% foliar injury in tobacco; 13% foliar injury in radish. 0-2% foliar injury in 4 species.				
same	0.05/0.10	0% foliar injury in 6 species	same	GH	+	same
same	0.10/0.10	1% foliar injury in tomato. 11-35% foliar injury in 5 species.	same	GH	+	
same	0.15/0.10	17-24% foliar injury in 6 species.	same	GH	+	same
same	0.05/0.20	6% foliar injury in soybean. 0-2% foliar injury in 4 species.	same	GH	+	same
same	0.20/0.20	0% foliar injury in tomato. 4-16% foliar injury in 5.				
same	0.05/0.25	7% foliar injury in soybean. 0-3% Foliar injury in 5 species.	same	GH	+	same
same	0.15/0.25	0-6% injury in 6 species.				
exposed continuously for 5 days a week. (103.5 hrs/wk for 20 wks)	0.11/0.11	72% reduction in leaf area. 83% reduction in dry weight of green leaves	Orchard grass (*Dactylis glomerata* var. Aberystwyth S37)	GH	+	Ashenden, 1979 1980
same	0.11/0.11	84% reduction in leaf area. 83% reduction in dry weight of green leaves	Kentucky bluegrass (*Poa pratensis* var. Monopoly)	GH	+	same
same	0.11/0.11	43% reduction in leaf area. 65% reduction in dry weight of green leaves	Italian ryegrass (*Lolium multiflorum* var. Milamo)	GH	+	same
same	0.11/0.11	52% reduction in leaf area. 84% reduction in dry weight of green leaves	Timothy (*Phleum pratense* var. Eskimo)	GH	+	same

[a] CE, Controlled environment; GH, greenhouse.

[b] +, Greater than additive; 0, additive.

Conversions (ppm to mg/m^3):

NO_2:	ppm	0.05	0.10	0.11	0.15	0.20	0.25
	mg/m^3	0.09	0.19	0.21	0.28	0.38	0.47
SO_2:	ppm	0.05	0.10	0.11	0.15	0.20	0.25
	mg/m^3	0.13	0.26	0.29	0.39	0.52	0.65

Source: OAQPS 1982

As noted in Chapter 2, NO_2 concentrations in clean, rural air are typically in the 1.88 $\mu g/m^3$ (0.001 ppm) range. This level is two orders of magnitude below the concentration required to produce 5 percent foliar injury (Table 4.3-4) even at 8 hours exposure.

Values for polluted air in urban centres in Sweden, Canada and the United States reported in Chapter 2 indicate NO_2 concentrations of 0.19 to 0.56 mg/m^3 (0.1 to 0.3 ppm) may occur for up to several hours. These concentrations are one order of magnitude lower than the concentrations known to cause 5 percent foliar injury levels, even with 8 hours exposure, to susceptible plant species, as summarized in Table 4.3-4 (Heck and Tingey, 1979). This would suggest that in most cases, significant injury or growth effects are unlikely to occur to urban ornamental plants, gardens or natural ecosystems.

It is possible that further research will develop more sensitive measures of vegetation response to NO_x concentrations, so that subtle effects on natural communities or other vegetation types may be identified. We currently see little evidence of such effects in the literature.

Combinations of NO_2 and SO_2 are known to reduce the injury threshold considerably, but unfortunately no method exists to make a quantitative assessment of these synergistic effects.

4.3.3 Photochemical Oxidants

Oxidant Effects on Vegetation

Recent studies have indicated that photochemical oxidants, primarily ozone, have a greater impact on agriculture and natural vegetation than do any other air pollutants (Heck _et_ _al_., 1977, 1980; Ormrod _et_ _al_., 1980; U.S. EPA, 1982a).

The three primary phytotoxic components of photochemical oxidants in polluted air include ozone (O_3), nitrogen dioxide (NO_2) and the peroxyacylnitrates (U.S. EPA, 1982a). NO_2 has been discussed separately. Since peroxyacetylnitrate (PAN) is the only member of the peroxyacylnitrate series usually detected in the atmosphere, it has received some attention in the literature, but the state of knowledge is much smaller for PAN than for O_3. Current research interest, and consequently, the contents of this review, are largely oriented towards the effects of O_3 on vegetation.

Natural Ecosystems and Forests

For the purpose of this discussion, natural ecosystems include terrestrial systems other than agricultural crops and ornamental gardens. While much of the research literature to date has focused upon agriculture, recent attention has now been directed towards questions related to O_3 effects on other vegetation communities (MOI, 1983; U.S. EPA, 1982a; Skelly, 1980; Smith, 1980).

In addition to direct injury or metabolic alteration to the plant itself, indirect alterations in other components or processes in the ecosystem due to O_3 exposure must be considered. The U.S. EPA (1982a) emphasized that disturbance from air pollutants is dose-related and that dose-response thresholds for a specific pollutant such as O_3 are very different among the various organisms comprising an ecosystem so that response of the ecosystem can be a very complex process. The authors noted that in repsonse to low doses of oxidants, the vegetation and soils function as a sink or receptor, but when exposed to intermediate doses, individual plant species or individual members of a given species may be subtly affected by nutrient stress, impaired metabolism, predisposition to insects or disease stresses or direct induction of disease. Exposure to high doses might induce acute morbidity or mortality of specific plants.

As noted by MOI (1983), there are differing considerations in evaluating the effects of O_3 on forest trees than for agricultural crops. Most forest tree species are long-lived perennial plants that are not subject to fertilization, soil amendments, cultivation, extensive pest control or other such practices. Their size to a great extent precludes pollutant exclusion (chamber) studies or protective sprays, so that the assessment of growth or productivity losses is limited to visual observation of growth characteristics, which must then be related to O_3 dose information if available.

Table 4.3-11 summarizes the effects of long-term, controlled exposure to ozone on growth, yield and foliar injury to several native plants and trees from a number of studies undertaken prior to 1978. Many North American species can be classified as susceptible to O_3 damage (Davis and Wilhour, 1976; Skelly, 1980) with direct injury to foliage being demonstrated in natural and experimental settings. Some authors suggest (Miller and McBride, 1975; Skelly, 1980) that existing concentrations of O_3 in some forested areas are sufficient to cause injury (MOI, 1983), and may affect the productivity, successional patterns and species composition of forests (Smith, 1980). Skelly (1980) summarized O_3 induced effects on temperate forest tree species and reported that oxidant air pollution may affect primary productivity, energy resource flow patterns, biogeochemical patterns and species successional patterns. The most important effects of O_3 on forest ecosystems (U.S. EPA, 1982a) include changes in succession, diversity and production, imposed through several mechanisms involving various components of the biota.

Primary producers (green plants) are sensitive to oxidant influences on reproduction, photosynthesis, respiration, other physiological processes, and foliar health (U.S EPA, 1982a). Consuming organisms are affected mainly through the food web, and studies to date have not indentified a direct impact of O_3 on these organisms. There is some indication that O_3 may enhance the development of disease caused by pathogens that normally infect stressed or senescent plant parts or invade nonliving woody plant tissues (Costonis and Sinclair, 1972; Weidensaul and Darling, 1979).

Clearly, the interactions between O_3 and various components of the natural ecosystem are complex and numerous. Case studies are currently

Table 4.3-11 EFFECTS OF LONG-TERM CONTROLLED OZONE EXPOSURES ON GROWTH, YIELD AND FOLIAR INJURY TO SELECTED PLANTS (USEPA 1978)

Plant species	Ozone Concentration µg/m^3 (ppm)	Exposure time, hr	Plant response percent reduction from control	Reference
Petunia	98-137 (0.05-0.07)	24/day, 53 days	30, flower fresh wt	Craker 1972
Poinsettia	196-235 (0.10-0.12)	6/day, 5 days/wk 10 wk	39, bract size	Craker & Feder 1972
E. white pine	196 (0.10)	4/day, 5 days/wk 4 wk (mixture of O_3 and SO_2 for same periods)	3, needle mottle (over 2-3 days of exposure)	Dochinger & Seliskar 1970
Carnation	98-177 (0.05-0.09)	24/day, 90 days	50, flowering (reduced vegetative growth)	Feder 1970
Geranium	137-196 (0.07-0.10)	9.5/day, 90 days	50, flowering (shorter flower lasting time, reduced vegetative growth)	
Lemna, duckweed	196 (0.10)	5/day, 14 days	100, flowering; 36, flowering (1 wk after exposure completed) 50, frond doubling rate	Feder & Sullivan 1969
Sweet corn cv. Golden Midget	98 (0.05)	6/day, 64 days	9, kernel dry wt; 14, injury (12, avg. 4 yield responses)	Heagle et al. 1972
	196 (0.10)	6/day, 64 days	45, 25, 35 for same responses	
Soybean cv. Dare	98 (0.05)	6/day, 133 days	3, seed yield; 22, plant fresh wt 19, injury, defoliation, no reduction in growth or yield	Heagle et al. 1974
	196 (0.10)	6/day, 133 days	55, 65, 36 for same responses	
Wheat	196 (0.10)	7/day, 54 days	16, yield	Heagle et al. 1979a
	255 (0.13)	7/day, 54 days	33, yield	
Field corn cv. Open-pedigree	294 (0.15)	7/day, 88 days	40, seed yield	Heagle et al. 1979b
cv. Coker 16	294 (0.15)	7/day, 88 days	12, seed yield	

Table 4.3-11 (Continued)

Plant species	Ozone Concentration $\mu g/m^3$ (ppm)	Exposure time, hr	Plant response percent reduction from control	Reference
Spinach	255 (0.13)	7/day, 38 days	69, fresh wt	Heagle et al. 1979c
	196 (0.1)	7/day, 38 days	37, fresh wt	
	117 (0.06)	7/day, 38 days	18, fresh wt	
Bean cv. Pinto	290 (0.15)	2/day, 63 days	33, plant dry wt; 46, pod fresh wt	Hoffman et al. 1973
	490 (0.25)	2/day, 63 days	95, plant dry wt; 99, pod fresh wt	
	686 (0.35)	2/day, 63 days	97, plant dry wt; 100, pod fresh wt	
Alfalfa	196 (0.10)	2/day, 21 days	16, top dry wt	Hoffman et al. 1974
	290 (0.15)	2/day, 21 days	26, top dry wt	
	390 (0.20)	2/day, 21 days	39, top dry wt	
Yellow poplar	588 (0.30)	8/day, 5 days/wk 13 wk	82, leaf drop; 0, height	Jensen 1973
Silver maple	588 (0.30)	8/day, 5 days/wk 13 wk	50, leaf drop; 78, height	
White ash	588 (0.30)	8/day, 5 days/wk 13 wk	66, leaf drop; 0, height	
Sycamore	588 (0.30)	8/day, 5 days/wk 13 wk	0, leaf drop; 22, height	
Sugar maple	588 (0.30)	8/day, 5 days/wk 13 wk	28, leaf drop; 64, height	
Poplar, hybrid	290 (0.15)	8/day, 5 days/wk 6 wk	50, shoot dry wt; 56, leaf dry wt 47, root dry wt	Jensen & Dochinger 1974
Wheat cv. Arthur 71	392 (0.20)	4/day, 7 days (anthesis)	30, yield	Kochhar 1974

Table 4.3-11 (Continued)

Plant species	Ozone Concentration µg/m^3 (ppm)	Exposure time, hr	Plant response percent reduction from control	Reference
Bean cv. Pinto	290 (0.15)	2/day, 14 days	8, leaf dry wt	Maas et al. 1973
	290 (0.15)	3/day, 14 days	8, leaf dry wt	
	290 (0.15)	4/day, 14 days	23, leaf dry wt (Data available on whole plants, roots, leaves, injury, and 3 levels of soil moisture stress)	
	290 (0.15)	6/day, 14 days	49, leaf dry wt	
	440 (0.225)	2/day, 14 days	44, leaf dry wt	
	440 (0.225)	4/day, 14 days	68, leaf dry wt (Data available on whole plants, roots, leaves, injury and 3 levels of soil moisture stress)	
	588 (0.30)	1/day, 14 days	40, leaf dry wt	
	558 (0.30)	3/day, 14 days	76, leaf dry wt	
Bean cv. Pinto	255 (0.13)	8/day, 28 days	79, top fresh wt 73, root fresh wt 70, height	Manning et al. 1971
Ponderosa pine	290 (0.15)	9/day, 10 days	4, photosynthesis	Miller et al. 1969
	290 (0.15)	9/day, 20 days	25, photosynthesis	
	290 (0.15)	9/day, 30 days	25, photosynthesis	
	290 (0.15)	9/day, 60 days	34, photosynthesis	
	588 (0.30)	9/day, 10 days	12, photosynthesis	
	588 (0.30)	9/day, 20 days	50, photosynthesis	
	588 (0.30)	9/day, 30 days	72, photosynthesis	
	880 (0.45)	9/day, 30 days	85, photosynthesis	
Alfalfa	196 (0.10)	6/day, 70 days	4, top dry wt, harvest 1 20, top dry wt, harvest 2 50, top dry wt, harvest 3	Neeley et al. 1977
	98 (0.05)	7/day, 68 days	30, top dry wt, harvest 1 50, top dry wt, harvest 2	
Beet, garden	392 (0.20)	3/day, 38 days	50, top dry wt	Ogata & Maas 1973
Sweet corn cv. Golden Jubilee	393 (0.20)	3/day, 3 days/wk until harvest	13, kernel dry wt; 20, top dry wt; 24, root dry wt	Oshima 1973
	686 (0.35)	3/day, 3 days/wk until harvest	20, kernel dry wt; 48, top dry wt; 54, root dry wt	

Table 4.3-II (Continued)

Plant species	Ozone Concentration $\mu g/m^3$ (ppm)	Exposure time, hr	Plant response percent reduction from control	Reference
Tomato	392 (0.20)	2.5/day, 3 days/wk 14 wk	1, yield; 32, top dry wt; 11, root dry wt	Oshima et al. 1975
	686 (0.35)	2.5/day, 3 days/wk 14 wk	45, yield; 72, top dry wt; 59, root dry wt	
Brome grass	290-647 (0.15-0.33) (Varied)	4/day, 5 days/wk growing season	83, biomass	Price & Treshow 1972
Radish	98 (0.5)	8/day, 5 days/wk 5 wk	54, root fresh wt 20, leaf fresh wt	Tingey et al. 1971
		8/day, 5 days/wk (mixture of O_3 and SO_2 for same periods)	63, root fresh wt 22, leaf fresh wt	
Soybean	98 (0.05)	8/day, 5 days/wk 3 wk	13, foliar injury	Tingey et al. 1973
		8/day, 5 days/wk (mixture of O_3 and SO_2 for same periods)	16, foliar injury; 20, root dry wt	
	196 (0.10)	8/day, 5 days/wk 3 wk	21, top dry wt; 9, root dry wt	
Alfalfa	98 (0.05)	8/day, 5 days/wk 12 wk	18, top dry wt	Tingey & Reinert 1975
Ponderosa pine	196 (0.10)	6/day, 126 days	12, root length 21, stem dry wt; 26, root dry wt	Wilhour & Neely 1977
W. white pine	196 (0.10)	6/day, 126 days	13, foliage dry wt; 9, stem dry wt	

Source: MOI, 1983

documenting evidence of O_3 influence on succession, species composition and productivity (Cobb and Stark, 1970; Miller, 1973; Miller et al., 1982; Hayes and Skelly, 1977). Figure 4.3-4 summarizes known effects of O_3 stress on a western coniferous forest ecosystem.

Hayes and Skelly (1977) speculated that susceptible eastern white pine (*Pinus strobus* L.) in certain areas may be rendered less competitive by O_3 stress, and other work (Skelly and Johnston, 1979; Skelly et al., 1982) has suggested shifts in species composition away from the importance of white pine, along with other changes in tree distribution, may be occurring in certain eastern regions (U.S. EPA, 1982a). Treshow and Stewart (1973) in community studies found that a single 2 hour exposure of aspen (*Populus tremuloides*) to 30 mg/m^3 (15 ppm) O_3 caused severe symptoms on 30 percent of the exposed foliage. Since white fir seedlings need aspen shade for best juvenile growth, this significant aspen loss may restrict white fir development and alter forest succession. Harward (1971) and Harward and Treshow (1975) concluded that only a year or two of plant exposure to O_3 levels above 0.14 to 0.60 mg/m^3 (0.07 to 0.3 ppm) may be enough to make major shifts in plant composition of understory components of forest ecosystems.

The U.S. EPA (1982a) reports that most woody plants susceptible to O_3 injury are generally species of an early successional stage (Harkov and Brennan, 1979), while most trees having intermediate or high tolerance are typically mid-or late-successional types. Mature ecosystems may be more resistant to air pollution stress.

Several studies have documented estimated reductions in forest production due to oxidant stress (Miller et al., 1977, 1982; Benoit et al., Mann et al., 1980). Figure 4.3-5 compares calculated growth of ponderosa pine in polluted (O_3) and non-polluted air (Miller and Elderman, 1977). An average 30-year-old tree grown in ambient air was estimated to reach a height of 7.0 m, to have a diameter of 19.0 cm and to be capable of producing one log 1.8 m long with a volume of 0.047 m^3 (U.S. EPA, 1982a). An average 30-year-old tree grown in the absence of oxidants was estimated to be 9.1 m in height, to have a diameter of 30.5 cm and to be capable of producing one log 4.9 m long with a volume of 0.286 m^3.

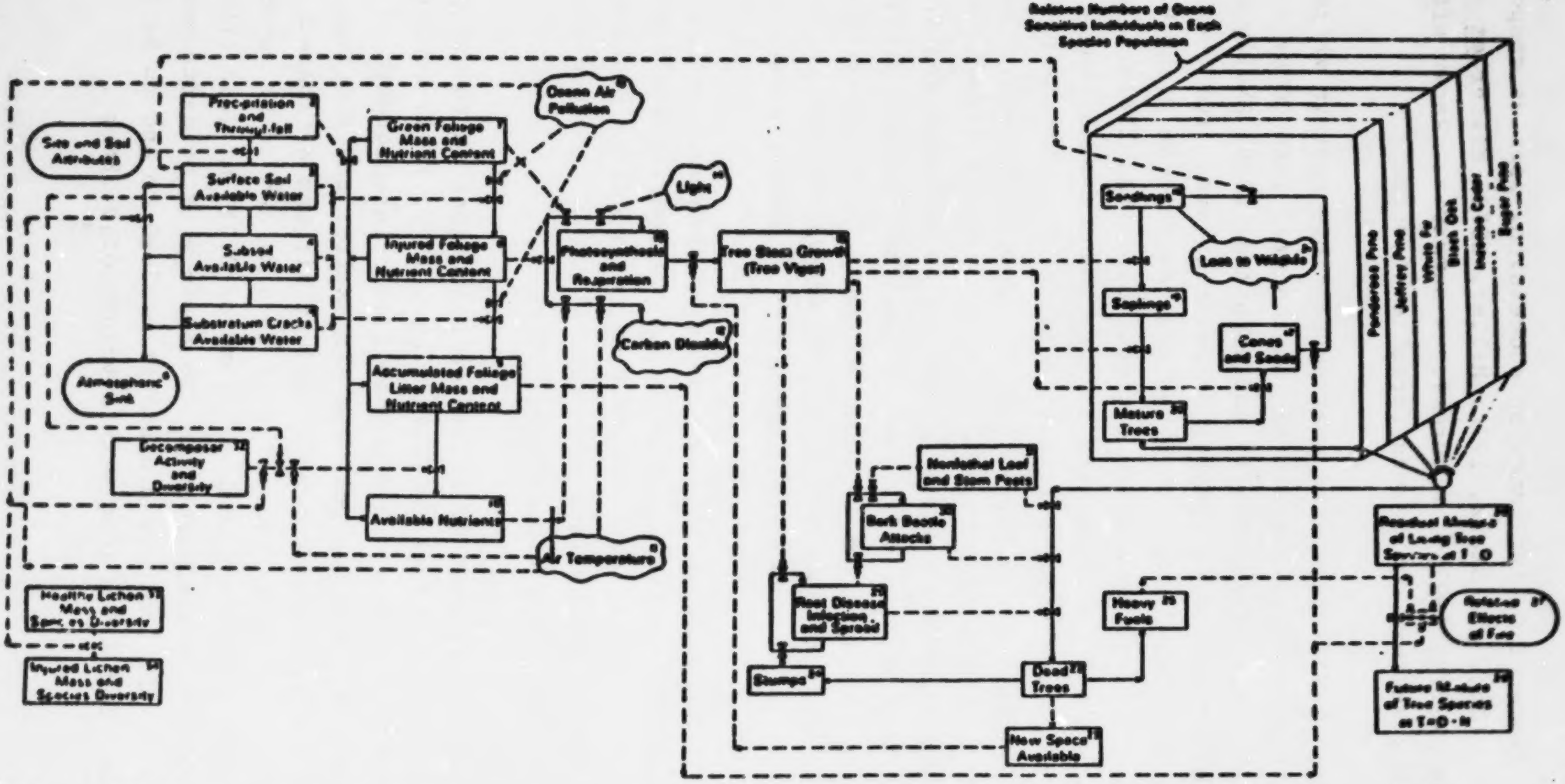

Ozone Air Pollution
Precipitation and Throughfall
Site and Soil Attributes
Surface Soil Available Water
Subsoil Available Water
Substratum Cracks Available Water
Atmospheric Sink
Decomposer Activity and Diversity
Green Foliage Mass and Nutrient Content
Injured Foliage Mass and Nutrient Content
Accumulated Foliage Litter Mass and Nutrient Content
Available Nutrients
Light
Photosynthesis and Respiration
Carbon Dioxide
Air Temperature
Healthy Lichen Mass and Species Diversity
Injured Lichen Mass and Species Diversity
Tree Stem Growth (Tree Vigor)
Relative Numbers of Ozone Sensitive Individuals in Each Species Population
Seedlings
Saplings
Mature Trees
Loss to Wildlife
Cones and Seeds
Ponderosa Pine
Jeffrey Pine
White Fir
Black Oak
Incense Cedar
Sugar Pine
Nonlethal Leaf and Stem Pests
Bark Beetle Attacks
Root Disease Infection, and Spread
Stumps
Dead Trees
Heavy Fuels
New Space Available
Resultant Mixture of Living Tree Species at T 0
Related Effects of Fire
Future Mixture of Tree Species at T=0+n

Figure 4.3-4 Diagrammatic model of the effects of chronic ozone air pollution stress on a western coniferous forest ecosystem. (from: Miller, et al. 1982).

Solid lines connecting boxes indicate material flow from compartment to compartment. Dashed lines indicate the controlling influence of one variable on the flow between compartments. The controlling "valve" resembles a bow tie.

Source: U.S. EPA, 1982 a

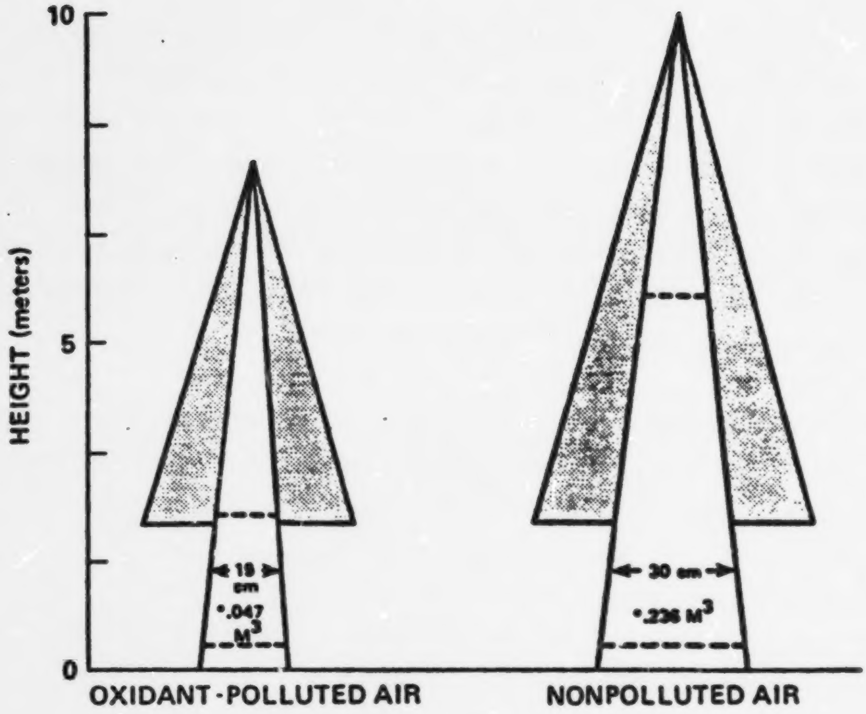

10
5
0
HEIGHT (meters)
19 cm
*.047 M^3
30 cm
*.236 M^3
OXIDANT-POLLUTED AIR
NONPOLLUTED AIR

Fig. 4.3-5 Calculated average growth of 30-year-old 15-cm San Bernardino National Forest, California, ponderosa pines in polluted and nonpolluted air based on radial growth samples from 1941-1971 and 1910-1940. The asterisk indicates wood volume in log with 15-cm top (min. merchantable diameter).

SOURCE: Miller and Elderman 1977.

Table 4.3-12 summarizes reported field observations of oxidant induced ecosystem perturbations. Data such as these have led to U.S. EPA (1982a) to conclude that large areas of the temperate forest ecosystem are currently experiencing perturbation from oxidant pollution. As well, the authors noted that the influence of ozone on patterns of succession and competition and on individual tree health is causing significant forest change in portions of the temperate zone. These changes are occurring in forest regions with ozone levels (1 hour maximum) ranging from 0.1 to 0.8 mg/m^3 (0.05 to 0.40 ppm).

The U.S. Environmental Protection Agency (1982a) has recognized oxidants as one of the most significant contemporary anthropogenic stresses imposed on temperate forest ecosystems. It notes that gradual and subtle changes in time in forest metabolism, growth and composition over wide areas of the temperate zone are the primary consequences of oxidant stress. Such changes were concluded to be much more important than the more dramatic destruction of forests in the immediate vicinity of point sources of pollution over short periods.

In terms of an estimate of effects of oxidants on Canadian natural ecosystems, few site data on ambient O_3 concentrations in non-urban settings are available, and detailed losses of forest productivity are lacking. A quantitative estimate of potential effects is therefore not possible at this time.

Agriculture: Extensive reviews of oxidant effects on agricultural crops have been compiled by U.S. EPA (1982a), Ormrod et al.(1980) and MOI (1983). Foliar responses of crops to artifical O_3 exposure have been widely documented and used in the development of species and varietal sensitivity listings and the preparation of dose-response curves (Larsen and Heck, 1976; Linzon et al., 1975). The recent Memorandum of Intent (1983) on Transboundary Air Pollution suggests that these data may not be reliable for estimating the total effect

Table 4.3-12 Field Observation of Oxidant Induced Ecosystem Perturbations.

Study		Pollutant				Ecosystem Perturbation		
			dose					
reference	location	oxidant	conc	time	analytical method	change in component(s)	change in structural pattern(s)	change in functional process(es)
McBride et al. 1975	San Bernardino Natl. Forest (SBNF), CA USA	ozone	3-4 pphm to 10-12 pphm	aver 24 hr May thru Sept. 1973-1978	NA			reduced production; ponderos pine
Gemmill 1980	SBNF	same	same	same	NA			reduced production; Californ black oak
Miller et al. 1980	SBNF	same	same	same	NA			reduced production; Californ black oak
Sigaland Nash 1983	SBNF	same	same	same	NA	reduced lichens	reduced diversity; 50% fewer species	
Miller & Eldermann 1977	SBNF	same	same	same		reduced primary producers; mainly ponderosa and Jeffrey pines; altered heterotrophs; foliar fungi, Fomes annosus, Dendroctonus brevicomis, tree squirrels	altered succession; from pine to oaks and shrubs	reduced production; ponderos pine, Jeffrey pine, white fi black oak altered biogeochemical cycli C,N,P,K,Ca,Mg,H_2O
Duchelle et al. 1983	Shenandoah Natl. Park, VA, USA	ozone	1979 4.7 pphm 8 pphm 1980 4.1 pphm 7 pphm 1981 3.6 pphm 7 pp	mean mntly. 8 hr. av. mean peak 1 hr. av. mean mntly. 8 hr. aver. mean mntly. 8 hr. av. mean peak 1	Bendix Model 8002 chemiluminescent O_3 analyzer			reduced production; native meadow vegetation

Table 4.3-12 (Cont'd)

Study reference		Pollutant dose				Ecosystem Perturbation		
reference	location	oxidant	conc	time	analytical method	change in component(s)	change in structural pattern(s)	change in functional process(es)
Benoit et al. 1982	Blue Ridge Mts. VA, USA	ozone	5-6 pphm	mnthly. 8 hr. av.Apr-Sept. 1979-1980	NA			reduced production; e. white pine
McLaughlin et al 1982	Cumberland Plateau,Oak Ridge,TN, USA	ozone	NA	NA	NA	reduced primary producer;sensitive 1. white pine		
Mann et al.1980	Cumberland Plateau, Oak Ridge, TN,USA	ozone	77 hr. ≧8 pphm	April-July 1976	NA			reduced production; sensitive e. white pine
			320 hr. ≧ 8 pphm	April-July 1977				

Conversions (pphm to µg/m^3)	pphm	3	3.6	4	4.1	4.7	5	6	7	8	12
	µg/m^3	60	72	80	82	94	100	120	140	160	240

Source: U.S. EPA 1982a

in crop productivity (yield, quality) since most information now indicates that the severity of foliar symptoms is not a reliable index of crop growth or yield effects (Reinert, 1980) as there is uneven competition among several sinks that receive photosynthate. Those studies also noted that compensatory responses to O_3 can produce rapid recovery from injury (Jacobson, 1982; Oshima et al., 1975; Tingey and Reinert, 1975) except where the harvested product is the foliage and where foliar injury development coincides with that rapid growth of the harvested product (Linzon et al., 1975).

Leaves are the primary receptors of photochemical oxidants in vascular plants (U.S. EPA, 1982a). To produce an effect in plants, oxidants must come into contact with a leaf, enter through the stomata into the air spaces, dissolve in the aqueous layer coating the cell walls, and pass through the membrane into the cell (Tingey, 1977). Cell membranes are considered to be the primary site of O_3 attack. Differential plant reponse has been related to environmental conditions, genetic expression, pollutant concentration, length of exposure, and the time elapsed between exposures.

The biochemical and physiological alterations caused by oxidants determine all the effects man considers important, including yield reduction and aesthetic loss (U.S. EPA, 1982a). The cellular responses of the plant are ultimately expressed as visible foliar injury, premature senescence, increased leaf abscission, reduced plant vigour, reduced growth and death. The oxidants of greatest phytotoxicological consequence are reported by those authors to be O_3 and PAN.

Ormrod et al. (1980) have summarized many of the documented effects of oxidants on Ontario crops, emphasizing foliar injury (Table 4.3-13). These include "weather fleck" of tobacco, "bronzing" of bean leaves, the "speckle-leaf" disorder of potato, "brown leaf" or oxidant stipple of grape leaves, "tip dieback" and leaf lesions of onions, and leaf injury due to PAN of tomatoes. Considerable oxidant-induced injury to crops was reported in southwestern Ontario and east of Toronto.

Table 4.3-13 A Summary of Reported Incidents of Photochemical Oxidant Injury to Agricultural Crops in Ontario

Cultivar	Year	Location	Leaf injury or rating (%)	Reference
Tobacco — weather fleck				
White gold	1959	Delhi	17.2†	Walker (1961)
White Gold	1960	Delhi	16.6–23.0†	Walker (1961)
White Gold	1959	Delhi	40‡	Walker and Vickery (1961)
Various	1956–61	Southern Ontario	Wide range	Macdowall et al. (1963)
White Gold	1960.61	Port Burwell	20§	Macdowall et al. (1964)
White Gold	1964(?)	Lake Erie shore	12§	Macdowall (1966)
Unspecified	1960.61	Lake Erie Shore	N.A.//	Cole and Katz (1966)
White Gold	1963	Delhi	3.6†	Walker (1967)
White Gold	1964	Delhi	10.2†	Walker (1967)
Unspecified	1972	South Ontario	$1 million¶	Gayed and Watson (1975)
Unspecified	1973	South Ontario	$1.35 million¶	Gayed and Watson (1975)
Delhi 34	1976	45 km N.E. Toronto	10.6	Bisessar and Temple (1977)
Hicks Broadleaf	1976	45 km N.E. Toronto	11.9††	Bisessar and Temple (1977)
Bean — bronzing				
Unspecified	1965	Erieau	60††	Weaver and Jackson (1968)
Clipper, Harkell	1967	Erieau	6‡‡	Weaver and Jackson (1968) Weaver et al. (1968) Haas (1970)
Sanilac	1973	Ridgetown	13§§	Curtis et al. (1975)
Potato — speckle leaf				
Irish Cobbler	1966,68	Harrow-Leamington	N.A.//	McKeen et al. (1973) Hooker et al. (1973)
Grape — brown leaf				
Various	1973	Vineland	Up to 100††	Kender and Carpenter (1974) Shuulis et al. (1972)
Onion — tip dieback				
Autumn Spice	1975	Bradford	28§§	Wukasch and Hofstra (1977a)
Autumn Spice	1975	Bradford	38////	Wukasch and Hofstra (1977b)
Tomato — PAN injury				
—	1972	S.W. Ontario	N.A.//	Pearson et al. (1974)
—	1973	Simcoe, Niagara	N.A.//	Pearson et al. (1974)
Celery — chlorosis and necrosis				
Tendercrisp	1974(?)	Port Colbourne	27.2††	Proctor and Ormrod (1977)

†Loss of value relative to leaves so injured as to be valueless.

‡With approx 5 cm irrigation per week.

§Fleck index (see references).

//Not assessed.

¶Loss estimate based on decrease in weight and quality of leaf and harvesting of immature leaves.

††Percent leaf area injured.

‡‡Maximum severity rating equals 9.

§§Percent yield increase using an ozone protectant.

////Percent yield increase in filtered air of an open-top chamber vs. unfiltered air.

Source: Ormrod *et al*, 1980

Pearson (1983) has summarized foliar assessment surveys for oxidant injury to field crops throughout southern Ontario for the period from 1971 to 1982. In that 12-year period, the Ontario government conducted 29 foliar injury assessment surveys of 9 sensitive crops on an annual or intermittent basis, comprising 1268 visual foliar injury ratings in 321 annually different crop varieties. These studies examined white beans, tomatoes, potatoes, tobacco and five muck crops.

For white beans, visual assessment surveys were conducted throughout the major areas of production in southern and southwestern Ontario to permit the comparison of the general severity of ozone injury to each year and to relate the degree of foliar injury to ozone episodes and other conditions known to govern the response of the crop to ozone exposure. Bronzing was most severe in 1976 and least in 1972 and 1971, while injury in 1982 and 1981 was found to be light. Pearson (1983) reported that in most years, injury severity was attributed to ozone episodes during the first 3 weeks of August, the time of maximum foliar sensitivity, Drought stress was found to have a significant protective effect.

These studies showed the following effects (Pearson, 1983) related to white beans:

1. The severity of bronzing on white beans was directly related to the chronological age and physiological development of the plants and coincided with the period of maximum foliar sensitivity during the first 3 weeks of August.
2. No one production area was more severely affected than any other during the survey years even when adjustments were made for differences in plant age and maturity.
3. Some slight differences in varietal sensitivity were detected but were found to be due to differences in plant age and thus did not represent a true genetic response.
4. The degree of root rot was found to fluctuate from one year to another and in the most severe case (1976) was associated with more severe foliar bronzing.

Pearson (1983) also noted that in spite of the absence of any quantitative information on the effect of ozone bronzing on crop yield, there has been evidence of major shifts in production areas, with the major trend being towards a decrease in Kent County and a shift to the northern counties of Huron and Perth. The author noted, however, that the yield of white beans has not totally influenced the growers' decision to increase or decrease their production acreage, and reported that available figures complicate the assessment of the effect of bronzing on the shift in bean production as it cannot be simplistically stated that the annual occurrence of the bronzing disorder in Kent and Elgin Counties has reduced bean yields resulting in a decrease in crop acreage. Nor is it true that bean production in the more northern counties increased because farmers were able to produce higher yields under bronze-free conditions even though there was no doubt that the bronzing syndrome has, in many years, significantly reduced bean yields and that bronzing has at times been more severe in the southern counties of Kent and Elgin (Pearson, 1983).

With respect to oxidant-related injury to tomatoes, referred to as PAN-type, studies reported by Pearson (1983) for southwestern Ontario from 1978 to 1981 led to the following conclusions:

1. Symptoms which appeared under field conditions were related to days of elevated ozone and plants could be protected against symptom development through the frequent use of an anti-ozonant protective chemical spray.
2. There was no apparent relationship between the development of the symptoms under field conditions and the presence of SO_2 and O_3 as suggested in several research reports dealing with synergism between these pollutants.
3. The symptoms which were atypical of those caused by ozone were reproduced under controlled environment conditions by a very narrow dose range involving ozone exposure.
4. Atmospheric PAN monitoring in 1980 and 1981 at Simcoe failed to confirm the presence of PAN in any significant quantities during periods when the PAN-type symptoms developed.

The annual tomato survey findings reviewed by Pearson (1983) revealed that since 1974, injury severity has varied considerably in intensity with differences also being detected in the geographical location of the most severely injured areas each year.

The extent and degree of upper and lower surface oxidant injury to potato foliage was assessed by the Ontario Ministry of the Environment throughout the major production areas of southern Ontario commencing in 1977 (Pearson, 1983). The foliar symptoms were reported to usually appear sometime between late June and mid-July when the plants have flowered and the tubers are developing. As the demands for photosynthetically produced nutrition at that time were at their peak, the potential for adverse yield effects was felt to be great.

Although no attempt had yet been made by Pearson (1983) or his colleagues to compare the severity from one region or year to another, the symptoms were observed in each year since 1977 and have ranged in severity from none to over 30%. Very noticeable varietal differences were observed and appeared to fit well with previously published sensitivity listings.

"Weather fleck" of tobacco was reported to range from less than 1% to 20% on flue-cured species and up to 8% on burley types (Pearson, 1983). The most severely affected areas were centred around Leamington and in the St. Thomas and Port Rowan areas.

Pearson (1983) also reported that in 1977, vegetable crops, including carrots, celery, lettuce, onions and radish, were examined for foliar ozone injury several times from June through August. In all cases, ozone injury to the various species was trace in severity, and appeared more pronounced during the later part of the growing season.

The flux of O_3 into the leaf appears to be influenced by stomatal aperture characteristics, stomatal density, genetic and environmental factors (Tingey and Taylor, 1982; Butler and Tibbitts, 1979a,b; Gesalman and Davis, 1978). Less is known about the uptake of PAN by plants (U.S. EPA, 1982a).

Once the oxidant passes into the liquid phase in the plant it may undergo chemical transformations which vary by plant species, ultimately eliciting biochemical and genetically influenced physiological responses. Studies have detected changes in proteins, fatty acids, sterols and sulfhydral residues as well as other biochemicals in plants exposed to PAN and O_3 (U.S. EPA 1982a).

Physiological responses to O_3 include changes in fluxes of carbohydrates, amino acids, inorganic ions and water (Heath, 1980; Tingey and Taylor, 1982) and once the oxidant has injured a cell membrane, many internal cellular responses occur (U.S. EPA, 1982a). These include metabolic pathways, membrane independent functions, and key steps in photosynthesis. In addition to depressing photosynthesis in the foliage of many plant species, O_3 inhibits the translocation of photosynthate from the top to roots (Tingey, 1974; Jacobson, 1982). The ultimate effect of these biochemical and physiological responses of plants to oxidants is a reduction in biomass and/or yield.

Recent reviewers (MOI, 1983; U.S. EPA, 1982a) have indicated that earlier workers studying oxidant effects emphasized foliar injury whereas more recently, the focus has shifted to effects on yields. An additional concern relates to the quality of that yield, including chemical composition, physical appearance and ability to be stored. The U.S. EPA (1982a) has noted, however, that although crop quality has important health and economic implications, it is difficult, at present, to completely correlate these O_3 effects with the more conventional measures of impacts on foliage and yield.

Several plant species have been found to be extremely sensitive biomonitors for detecting the presence of O_3, including the tobacco cultivar Nicotiana tabacum cv. Bel-W3, bean cultivars (Phaseolus vulgaris L.), soybean (Glycine max L.), tomato (Lycopersicon exculentum L.), begonia (Begonia semperflorens, Link and Otto), petunia (Petunia hybrida, Vilm.), morning glory (Pharbitis nil var. Scarlet O'Hara), and common milkweed (Asclepias syriaca L.) (U.S. EPA, 1982a).

Earlier attempts to develop exposure-response relationships in the United States (U.S EPA, 1978) were based primarily on foliar injury, as were the 1976 O_3 Criteria in Canada (Fisheries and Environment Canada, 1976), since models relating a given dose to a meaningful plant effect (yield) were not then available.

The American values representing the lowest concentration and duration of exposure that resulted in foliar injury from O_3 and PAN were as follows (U.S. EPA, 1978):

The limiting values for O_3 effects on agricultural crops were:

0.5 hr: 0.4 to 0.8 mg/m^3 (0.2 to 0.4 ppm)
1 hr: 0.2 to 0.5 mg/m^3 (0.1 to 0.25 ppm)
4 hr: 0.08 to 0.18 mg/m^3 (0.04 to 0.09 ppm)

For PAN effects on agricultural crops, the limiting values were:

0.5 hr: 0.4 mg/m^3 (0.2 ppm)
1 hr: 0.2 mg/m^3 (0.1 ppm)
4 hr: 0.07 mg/m^3 (0.035 ppm)

The U.S. EPA (1978) also produced an estimate of 0.1 to 0.2 mg/m^3 (0.05 to 0.10 ppm) O_3 as a threshold value for measurable growth effects on sensitive cultivars exposed for a few hours per day for several days, weeks, or months. That agency (U.S. EPA, 1982a) noted that superimposing O_3 additions to the ambient load in open-top chamber under simulated normal agronomic practices is probably generating the best exposure response data to date. The current ambient concentrations are having a significant impact on the yield of some crops. Characterizing dose as a single statistic representing the 7-hour midday seasonal average O_3 concentration provides a range of 0.084 to 0.200 mg/m^3 (0.042 to 0.100 ppm) for significant effects on sensitive plant species.

Certain recent studies have generated dose-response functions for O_3 by simulating normal exposure dynamics by exposing crops for 7-hours each day over a growing season to several concentrations of O_3 superimposed on the ambient O_3 load (Heagle et al., 1979; Heck et al., 1982), as part of the National Crop Loss Assessment Network (NCLAN). A general sensitivity ranking of studied crops (U.S. EPA 1982a) would show turnip, lettuce, peanut, spinach, and soybean as O_3 - sensitive (0.084 to 0.120 mg/m^3) and likely to be suffering significant losses, while wheat and kidney bean are intermediate (threshold between 0.12 and 0.20 mg/m^3). The authors noted that the data suggest ambient O_3 levels in many different parts of the U.S. are capable of causing significant reductions in the growth and yield of many different species. Significant growth reductions were noted in response to seasonal daytime average O_3 concentrations as low as 0.084 to 0.12 mg/m^3 (0.042 to 0.06 ppm).

Tables 4.3-14 and 4.3-15 summarize effects of chronic exposures to O_3 in closed chambers on growth and yield of selected crops and forest trees, respectively. Table 4.3-16 draws together models describing the relationship between foliar injury and O_3 exposure, while Table 4.3-17 is a summary of models of crop yields and losses due to O_3 exposure.

Teng et al.(1982) described crop-loss models which used several independent variables representing periods of exposure to O_3 within a season (Table 4.3-18). This and other predictive modelling approaches, although promising, have yet to be validated with commercial yield plots.

Table 4.3-19 summarizes pollutant dose-crop response functions developed during 1981 NCLAN studies as reported by Heck et al., (1982a). Table 4.3-20 reports percent yield reduction as a function of O_3 concentration for a wide range of crop types (Heck et al., 1982b), while Table 4.3-21 lists crop loss functions for four major species and an "all other" category as a function of O_3 dose.

Table 4.3-14 EFFECTS OF CHRONIC EXPOSURES TO OZONE IN CLOSED CHAMBERS ON THE GROWTH AND YIELD OF SELECTED CROPS

Plant species	O_3 concentration (ppm[a])	O_3 Dose	Exposure time,	Plant response, percent change from control (negative change unless otherwise noted)	Reference
Red kidney beans	0.016 (peak hr avg.	10.12 ppm)	71 d	Control, 90, charcoal-filtered air, Riverside, CA	Oshima, 1978
	0.074 (peak hr avg.	27.94 ppm)	71 d	+8, seed wt	
	0.148 (peak hr avg.	51.61 ppm)	71 d	+2, seed wt	
	0.221 (peak hr avg.	69.02 ppm)	71 d	65, seed wt	
	0.295 (peak hr avg.	83.61 ppm)	71 d	78, seed wt	
Potato	0.03 (peak hr avg.	4.98 ppm)	106 d	Control, 90, charcoal-filtered air, Riverside, CA	Foster, 1980
	0.08 (peak hr avg.	16.88 ppm)	106 d	35, no. of tubers; 30, tuber wt	
	0.18 (peak hr avg.	29.54 ppm)	106 d	25, no. of tubers; 16, tuber wt	
	0.27 (peak hr avg.	41.40 ppm)	106 d	37, no. of tubers; 42, tuber wt	
Soybean (4 cultivars)	0.03		6 hr/d, 10 d	2, shoot fresh wt; 0, root fresh wt	Heagle, 1979b
	0.06		6 hr/d, 10 d	2, shoot fresh wt; +2, root fresh wt	
	0.09		6 hr/d, 10 d	4, shoot fresh wt; 4, root fresh wt	
	0.12		6 hr/d, 10 d	15, shoot fresh wt; 16, root fresh wt	
	0.15		6 hr/d, 10 d	16, shoot fresh wt; 24, root fresh wt	
Pepper	0.12		3 hr/d, 3 d/wk, 11 wk	19, dry wt fruit	Bennett et al., 1979
	0.20			50, dry wt fruit	
Turfgrass (18 cultivars)	0.15		6 hr/d, 10 d	8, total leaf area (range 27 to +38)	Elkiey and Ormrod 1980
Potato (Norland)	0.20		3 hr/2 wk, 17-20 wk	20, no. of tubers; 25, tuber wt; 13 total solids in tubers	Pell et al., 1980
(Kennebec)	0.20			36, no. of tubers; 42, tuber wt; 20, total solids in tubers	
Cotton	0.25		6 hr/d, 2 d/wk, 13 wk	52, no. of bolls; 62, dry wt ginned fiber	Oshima et al., 19
	0.25		6 hr/d, 2 d/wk, 18 wk	55, no. of bolls; 59, dry wt ginned fiber	
Soybean	0.25		4 hr/d, 3 d/wk, 5 wk	44, plant dry wt; 66, no. of nodules	Reinert and Weber, 1980
	0.25		4 hr/d, 3 d/wk, 7 wk	61, plant dry wt; 69, no. of nodules	
	0.25		4 hr/d, 3 d/wk, 9 wk	43, plant dry wt; 49, no. of nodules	
	0.25		4 hr/d, 3 d/wk, 11 wk	46, plant dry wt; 46, no. of nodules	
Radish	0.3		3 hr, 9 times in 3 wk	96, root fresh wt	Reinert and Sanders, 1982
Marigold	0.3		3 hr, 9 times in 3 wk	20, flower dry wt	Reinert and Sanders, 1982

[a] 1 ppm O_3 = 1960 $\mu g/m^3$.

Source: U.S. EPA 1982a

Table 4.3-15 EFFECTS OF CHRONIC EXPOSURES TO OZONE IN CLOSED CHAMBERS ON SELECTED FOREST TREE SPECIES

Plant species	O_3 concentration (ppm^a)	Exposure time	Plant response, percent change from control (negative change unless otherwise noted)	Reference
Poplar (2 cultivars)	0.041	12 hr/d, 5 mo	8, stem dry wt; 61, no. of leaves on stem; +1013, no. of dropped leaves; 3, total dry wt	Mooi, 1980
Loblolly pine (2 families)	0.05	6 hr/d, 28 d	5, height growth	Kress et al., 19
American sycamore (2 families)	0.05	6 hr/d, 28 d	5, height growth	Kress et al., 198
Loblolly pine	0.05 0.10 0.15	6 hr/d, 28 d	18, height growth; 14, total dry wt 27, height growth; 22, total dry wt 41, height growth; 28, total dry wt	Kress and Skelly, 1982
Pitch pine	0.05 0.10 0.15	6 hr/d, 28 d	4, height growth; 8, total dry wt 13, height growth; 19, total dry wt 26, height growth; 24, total dry wt	Kress and Skelly, 1982
Virginia pine	0.05 0.10 0.15	6 hr/d, 28 d	5, height growth; +2, total dry wt 11, height growth; 3, total dry wt 14, height growth; 13, total dry wt	Kress and Skelly, 1982
Sweetgum	0.05 0.10 0.15	6 hr/d, 28 d	+9, height growth; 10, total dry wt 29, height growth; 26, total dry wt 45, height growth; 42, total dry wt	Kress and Skelly, 1982
American sycamore	0.05 0.10 0.15	6 hr/d, 28 d	+4, height growth; 23, total dry wt 27, height growth; 61, total dry wt 21, height growth; 69, total dry wt	Kress and Skelly, 1982
White ash	0.05 0.10 0.15	6 hr/d, 28 d	+12, height growth; +22, total dry wt 9, height growth; 9, total dry wt 15, height growth; 17, total dry wt	Kress and Skelly 1982

Table 4.3-15 (Continued)

Plant species	O_3 concentration (ppm[a])	Exposure time	Plant response, percent change from control (negative change unless otherwise noted)	Reference
Green ash	0.05	6 hr/d, 28 d	2, height growth; 14, total dry wt	Kress and Skelly, 1982
	0.10		24, height growth; 28, total dry wt	
	0.15		30, height growth; 33, total dry wt	
Willow oak	0.05	6 hr/d, 28 d	1, height growth; 2, total dry wt	Kress and Skelly, 1982
	0.10		4, height growth; 11, total dry wt	
	0.15		19, height growth; 13, total dry wt	
Sugar maple	0.05	6 hr/d, 28 d	5, height growth; 3, total dry wt	Kress and Skelly, 1982
	0.10		+8, height growth; 7, total dry wt	
	0.15		12, height growth; 41, total dry wt	
Yellow poplar	0.05	6 hr/d, 28 d	+60, height growth; +41, total dry wt	Kress and Skelly, 1982
	0.10		+8, height growth; +5, total dry wt	
	0.15		12, height growth; +18, total dry wt	
Yellow poplar	0.10	12 hr/d, 50 d	19, relative growth rate; 16, net assimilation rate; 37, relative leaf area growth rate	Jensen, 1981a
Cottonwood	0.10	12 hr/d, 50 d	59, relative growth rate; 58, net assimilation rate; 52, relative leaf area growth rate	Jensen, 1981a
White ash	0.10	12 hr/d, 50 d	No significant effects	Jenson, 1981a
White ash	0.1	4 hr/d, 1 d/wk, 9 wk	+13, total height; +7, shoot dry wt	McClenahen, 1979
	0.2		0, total height; +5, shoot dry wt	
	0.3		0, total height; 11, shoot dry wt	
	0.4		0, total height; 14, shoot dry wt	
Black cherry	0.1	4 hr/d, 1 d/wk, 9 wk	+16, total height; +15, shoot dry wt	McClenahen, 1979
	0.2		+5, total height; 4, shoot dry wt	
	0.3		+3, total height; 4, shoot dry wt	
	0.4		28, total height; 15, shoot dry wt	
Hybrid poplar	0.2	5 hr/d, 5 times in 1 mo	37, total dry wt; 8, height growth	Harkov and Brennan, 1982
Hybrid poplar	0.2	7.5 hr/d, 5 d/wk, 6 wk	5, height; +2, leaf dry wt; 6, chlorophyll content	Jensen, 1979
	0.2 (0.1 to 0.3)	7.5 hr/d, 5 d/wk, 6 wk	8, height; 13, leaf dry wt; +224, chlorophyll content	
Hybrid poplar	0.25	12 hr/d, 24 d	50, no. of leaves; 65, leaf area; 66, leaf dry wt	Noble and Jensen, 1980
Green ash	0.5	8 hr/d, 5 d/wk, 2 wk	10, stem dry wt; +3 leaf dry wt; 14, root starch	Jensen, 1981b
		8 hr/d, 5 d/wk, 4 wk	46, stem dry wt; 21, leaf dry wt; 17, root starch	
		8 hr/d, 5 d/wk, 6 wk	43, stem dry wt; 32, leaf dry wt; 23, root starch	

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[a] 1 ppm = 1960 μg/m^3.   Source: U.S. EPA 1982a

Table 4.3-16 SUMMARY OF MODELS DESCRIBING THE RELATIONSHIP BETWEEN FOLIAR INJURY AND OZONE EXPOSURE

Model	Plant species	Reference
1. $y = a + bx$ y = injured leaves, area (%) x = ozone index (ppb x hr)[a] a = -3.5 (winter), -0.38 (summer), -1.85 (fall) b = 0.0037 (winter), 0.0016 (summer), 0.0015 (fall)	Tobacco Bel-W3	Goren and Donagi, 1980
2. $y = a + bx$ y = probit of % leaves with injury x = log of ozone dose (pphm-hr) based on 20 pphm concentration a = 4.3 b = 1.3 LD_{50} = dose injuring 50% of leaves 372 (pphm-hr)	Tobacco White Gold	MacDowall and Cole, 1971
3. $P = P_k(1-e^{-kt})$ P = % injured leaves at time t P_k = equilibrium % of injured leaves k = constant determined by least squares	Tobacco Bel-W3	Naveh and Chaim, 1978
4. $C = A_0 + A_1I + A_2/t$ C = ozone concentration A_0, A_1, A_2 = regression coefficients I = percent foliar injury t = time of exposure	Selected species	Heck and Tingey, 1971

Table 4.3-16 (continued)

Model	Plant species	Reference
5. $Z = -\ln Mghr/\ln Sg - p \ln t/\ln Sg + \ln C/\ln Sg$ Z = no. of standard deviations that the percentage of injury is from the median C = ozone concentration t = exposure duration Mghr = geometric mean concentration Sg = standard geometric deviation p = slope of the line on logarithmic paper.	Selected species	Larsen and Heck, 1976
6. Model 5 $\text{Probit } (y) = 1.3 \ln c + 0.49 \ln d + 0.77$ where c = concentration in µl/l d = duration in hrs y = % leaf surface injured	Soybean cv. Hodgson	Pratt and Krupa, 1981
7. Model 5 $PIF = 0.2174 + 2.2457 \ln c + 2.1378 \ln t$ where c = concentration in µl/l t = duration in hrs PIF = Probit mean proportion of injured foliage/plant	Black cherry	Davis et al., 1981
8. Short-term controlled fumigations $S = n \ln D + K$ where $D = (C^{m/n} \times t)$ and S is in the range 0 to 1 S = plant injury degree^{b} C = concentration in ppm t = exposure duration in hrs m = constant n = constant K = constant $S = 0.278 \ln D + 0.999$	Morning glory	Nouchi and Aoki, 1979

Table 4.3-16 (continued)

Model	Plant species	Reference
9. Ambient conditions $S = n \ln D + A \ln D' + K'$ where $D = \Sigma_i C_i^{m/n}$ S = plant injury degree[b] C_i = hourly average concentration at the ith hour in ppm $A \ln D'$ = contribution to the injury on the day due to the effects of oxidant dosage up to the previous day A = constant K' = constant $S = 0.278 \ln D_j + 0.041 (\ln D_{j-1} + \ln D_{j-2} + \ln D_{j-3}) + 1.872$	Morning glory	Nouchi and Aoki, 1979

[a] half the accumlating sum of average hourly ozone concentration between the first value $\geq$ 40 ppb and the last value < 40 ppb.

[b] plant injury degree = (Σ% damaged leaf area per leaf)/Σ area of the leaves that can be damaged to the maximum degree.

Source: U.S. EPA 1982a

Table 4.3-17 SUMMARY OF MODELS OF OZONE YIELD AND LOSS

Model	Crop	Reference
1. a) Total fresh wt. function $y = a + bx$ y = fresh wt (g/plant) $y = 162.4 - 0.015x$ a = intercept x = ozone dose (pphm-hr > 10 pphm)	Alfalfa cv Moapa 69	Oshima et al., 1976
b) Loss function - transformed from 1a by % loss = $(a - wt)/a \times 100$ % Loss = $-1.068 \times 10^{-4} + 9.258 \times 10^{-3}x$ x = ozone dose (pphm-hr > 10 pphm)		
2. a) Marketable fruit $y = [\sin(-0.0076x + 84.2816)]^2$ x = % fruit marketable USDA minimum size x = ozone dose (pphm-hr > 10 pphm)	Tomato VF 6718	Oshima et al., 1977
b) Yield function $y = 9.742 - 0.0023x$ y = container yield based on USDA fruit size and packing configuration x = ozone dose (pphm-hr > 10 pphm)		
c) Loss function - transformed from 1b by % loss = (a - container yield)/a × 100 % Loss = $0 + 0.0232x$ x = ozone dose (pphm-hr > 10 pphm)		
3. a) $y = a + bx$ y = yield (varies with crop) x = ozone exposure in seasonal 7 hr/d mean ozone concentration (ppm) a = intercept b = slope	Selected crops (Field corn, winter wheat, soybeans, spinach)	Heagle and Heck, 1980
b) $y = a + b_0x + b_1x^2$ y = yield (varies with crop) x = ozone exposure in seasonal 7 hr/d mean ozone concentration (ppm) a = intercept b_0 and b_1 = regression coefficients		

Table 4.3-17 (continued)

Model	Crop	Reference
4. a) Linear yield function $y = b_0 + b_1x$ y = crop yield (g/plant) x = ozone exposure in seasonal 7 hr/d mean concentration (ppm) b_0 = intercept b_1 = slope	Selected crops	Heck et al., 1982
b) Plateau - linear yield function $y = b_0$ if $x \leq f$ $y = (b_0 - b_1f) + b_1x$ if $x > f$ y = crop yield (g/plant) x = ozone exposure in seasonal 7 hr/d mean concentration (ppm) f = threshold 7 hr/d mean concentration (ppm) b_0 = intercept b_1 = slope		
c) Loss function $y = \frac{100}{a} b_1 (0.025 - x)$ y = % yield reduction b_1 = regression coefficient from function 4a a = predicted yield from function 4a at 0.025 ppm 7 hr/d mean ozone concentration in g/plant x = ozone exposure in seasonal 7 hr/d mean ozone concentration		
5. a) Tuber weight yield function $y = a + bx$ $y = 1530 - 15.8D$ y = % tuber yield in g/plant D = ozone dose in ppm-hr	Potato cv Centennial Russet	Foster et al., 1982
b) Tuber number yield function $y = 34.3 - 0.318D$ y = tuber yield in number/plant D = ozone dose in ppm-hr		
c) Plant dry matter function $DM = 382 - 3.83D$ DM = total dry matter in g/plant D = ozone dose in ppm-hr		

Source: U.S. EPA, 1982a

Table 4.3-18 SUMMARY OF CROP-LOSS MODELS

	Model	Loss criteria
General Model		
1. $y = f(x_{t_i})...$	y = proportion of yield reduction x_i = dose parameter at time t_i	NA
2. net yield reduction is	$\sum_{i}^{n} ydt$ dt = time step n = maximum number of growing days	NA
Functional Models		
1. Alfalfa		
$y = ax + bx^2 + cx^3$	y = daily yield loss (Fresh wt) $x = \Sigma$ hourly averages for 1 d a to c = regression coefficients	$\text{Loss} = 1.0 - \frac{\text{Biomass at site (x)}}{\text{Biomass at control site}}$
2. Corn		
$y = ax_1 + bx_2....1x_{12}$	y = yield loss based on 100 kernel wt x_1 to x_{12} = ozone summary statistic for periods 1 to 12 calculated as: $\frac{\sum_{i=1}^{N} [(\Sigma hi/n)24]}{N}$	$\text{Loss} - 10 - \frac{\text{100 kernel yield for (x)}}{\text{100 kernel yield for control treatment}}$

Table 4.3-18 (continued)

Model		Loss criteria
	where: N is the number of days in a period (7) hi = ozone concentrations n = number of hours for which there are ozone concentrations a to l = regression coefficients	
3. Wheat		
$y = ax_1 + bx_2 \ldots gx_7$	y = yield loss based on 100 seed x_1 to x_7 = ozone summary statistics for periods 1 to 7 calculated as: $\frac{\sum_{i=1}^{N} [(\Sigma hi/n)24]}{N}$ where: N is the number of days in a period (7) hi = ozone concentrations n = number of hours for which there are ozone concentrations a to g = regression coefficients	Loss = 1.0 - $\frac{\text{100 seed yield for (x)}}{\text{100 seed yield for carbon filtered treatment}}$
4. Potato		
$y = ax_1 + bx_2 + cx_3 + dx_4 + ex_5$	y = yield loss based on tuber wt/plant x_1 to x_5 = ozone summary statistic for periods 1 to 5 calculated as: $\frac{\sum_{i=1}^{N} [(\Sigma hi)]}{N}$ Where: N is the number of days in a period (14) hi = hourly ozone concentrations in 1 d a to e = regression coefficients	Loss = 1.0 - $\frac{\text{tuber wt yield for (x)}}{\text{tuber wt yield for control treatment}}$

Source: Teng et al., 1982.

Source: U.S. EPA, 1982a

Table.4.3-19 SUMMARY OF POLLUTANT DOSE-CROP RESPONSE FUNCTIONS DEVELOPED IN 1981

Pollutants	Crop/cv.	Location	Yield Response to Pollutants (kg/ha)
Ozone*	Corn/ Pioneer 3780	Argonne, Illinois	$y = 10,836 + t(-78,993 [O_3 - 0.071])$ If $O_3 < 0.071$, $t = 0$ If $O_3 > 0.071$, $t = 1$
	PAG 397		$y = 12,221 + t(-105,751 [O_3 - 0.090])$ If $O_3 < 0.090$, $t = 0$ If $O_3 > 0.090$, $t = 1$
	Soybean/ Hodgson	Ithaca, New York	$y = 2628 - 9875 (O_3)$
	Davis	Raleigh,	$y = 5,345 - 39,886 (O_3) + 109,600 (O_3)^2$
	Williams	Beltsville, Maryland	$y = 4,426 - 110,429 (O_3)$
	Essex		$y = 3,901 - 5,038 (O_3)$
	Cotton Lint/ Acala SJ2	Shafter, California	Normal $y = 2036 - 6884 (O_3)$ Water-stressed $y = 1301 - 2784 (O_3)$
Ozone/SO_2	Soybean/ Davis	Raleigh, North Carolina	For $SO_2 = 0.026$ ppm $y = 5220 - 39194 (O_3) + 109,600 (O_3)^2$ for $SO_2 = 0.085$ ppm $y = 4937 - 37624 (O_3) + 109,600 (O_3)^2$ for $SO_2 = 0.367$ ppm $y = 3585 - 30120 (O_3) + 109,600 (O_3)^2$
	Williams & Essex#	Beltsville, Maryland	For $SO_2 = 0.071$ ppm $y = 4,503 - 32,798 (O_3) + 164,897 (O_3)^2$ For $SO_2 = 0.148$ ppm $y = 4,212 - 25,322 (O_3) + 103,541 (O_3)^2$ For $SO_2 = 0.334$ ppm $y = 3,863 - 26,153 (O_3) + 92,033 (O_3)^2$

* Ozone seasonal 7-hr average concentrations in ppm.

# Combined data.

Source: Heck, _et al_, 1982a

Table 4.3-20 **Percent yield reduction as a function of O_3 concentration.**[a]

Crop	Linear function[b]	Predicted % yield reduction at two O_3 conc.[d] (±SE) 0.06 ppm	0.10 ppm
Corn (Coker 16)	$Y = -2.7 + 108\,x$	3.83 ± 1.0	8.1 ± 2.1
Soybean (Corsoy)	$Y = -15.5 + 621\,x$	21.7 ± 1.4	46.5 ± 2.9
Soybean—1977 (Davis)	$Y = -11.1 + 443\,x$	15.5 ± 2.4	33.2 ± 5.1
Soybean—1978 (Davis)	$Y = -8.8 + 353\,x$	12.3 ± 3.6	26.4 ± 7.7
Kidney Bean (Calif. Light Red)	$Y = -4.8 + 193\,x$	6.8 ± 1.3	14.5 ± 2.8
Lettuce, Head (Empire)	$Y = -16.3 + 652\,x$	22.8 ± 1.7	48.9 ± 3.6
Peanut (NC-6)	$Y = -17.8 \pm 711\,x^c$	24.9 ± 0.7	53.3 ± 1.5
Spinach (America)	$Y = -13.2 + 527\,x$	18.5 ± 3.0	39.6 ± 6.4
Spinach (Hybrid 7)	$Y = -13.0 + 517\,x$	18.1 ± 3.7	38.9 ± 8.0
Spinach (Viroflay)	$Y = -14.8 + 594\,x$	20.7 ± 3.1	44.4 ± 6.6
Spinach (Winter Bloomsdale)	$Y = -14.9 + 599\,x$	20.9 ± 3.4	44.8 ± 7.3
Turnip (Just Right)	$Y = -22.1 + 887\,x^c$	31.0 ± 2.1	66.4 ± 4.5
Turnip (Purple Top White Globe)	$Y = -20.5 + 817\,x^c$	28.6 ± 2.4	61.4 ± 5.0
Turnip (Shogoin)	$Y = -20.5 + 818\,x$	28.6 ± 2.6	61.4 ± 5.5
Turnip (Tokyo Cross)	$Y = -19.1 + 763\,x^c$	26.8 ± 4.6	57.4 ± 9.8
Wheat (Blueboy II)	$Y = -7.4 + 290\,x$	10.4 ± 1.4	22.3 ± 3.0
Wheat (Coker 47-27)	$Y = -10.1 + 405\,x$	14.2 ± 1.2	30.4 ± 2.6
Wheat (Holly)	$Y = -7.5 + 304\,x^c$	10.6 ± 1.5	22.6 ± 3.3
Wheat (Oasis)	$Y = -6.2 + 250\,x$	8.7 ± 1.7	18.6 ± 3.7

[a] This includes all data sets. The O_3 is expressed as the seasonal 7-h/day mean concentration.

[b] $Y = \frac{100\,b_1}{a}[0.025 - x]$

where Y = predicted percent yield reduction, b_1' = the regression coefficient from the yield model, a = the predicted yield at a seasonal 7-h/day mean O_3 concentration of 0.025 ppm and x = seasonal 7-h/day mean O_3 concentration.

[c] These models must be considered with caution. More complex models account for significantly more of the variation than these simple linear models. Compare the predicted percent yield reductions for the plateau-linear model at 0.06 ppm to get an idea of the differences.

[d] Values may differ slightly from those predicted by the equations due to a rounding of numbers in the functions shown. The 0.06 ppm seasonal 7-h/day mean O_3 concentration is the maximum mean concentration expected in many parts of the U.S. when the one-hr standard of 0.12 ppm is just met; the 0.10 ppm was arbitrarily used as a maximum 7-h/day mean O_3 concentration for comparison.

Source: Heck et al, 1982b

Table. 4.3-21 Crop loss functions for four major species and an "all other" category as a function of O_3 dose.[a]

Crops	Linear model	Predicted % yield reduction at seasonal 7-h/day mean O_3 concentrations[d]		
		0.06	0.08	0.10
Soybean[b]	$Y = -11.8 + 472x$	16.5	26.0	35.4
Winter Wheat[b]	$Y = -7.8 + 313x$	11.0	17.2	23.5
Corn	$Y = -2.7 + 108x$	3.8	5.9	8.1
Peanut	$Y = -17.8 + 711x$	24.9	39.1	53.3
All Other[b,c]	$Y = -13.0 + 519x$	18.1	28.5	38.9

[a] All equations give yield (Y) as a % reduction; they were developed from data in Table V.

[b] There is no real statistical legitimacy for these combination models. This is just an averaging of a number of heterogeneous data from Table VII. However, the technique permits the development of reasonable yield reduction models that approximate the change in yield as a function of O_3 dose.

[c] This model is only a guide in estimating crop losses for all species not specifically listed. It is intended for use with broad groups of crops and should not be used for estimating losses to any single species (e.g., radish or carrot).

[d] Values may differ slightly from those predicted by the equation due to a rounding of numbers in the functions shown. The 0.06 ppm seasonal 7-h/day mean O_3 concentration is the maximum mean concentration expected in many parts of the U.S. when the one-hour standard of 0.12 ppm is just met. The 0.08 ppm seasonal mean O_3 concentration would be found in some areas of the U.S. where the one hour standard of 0.12 ppm is often exceeded. The 0.10 ppm seasonal mean O_3 concentration was arbitrarily used as a maximum concentration for comparison.

Source: Heck et al, 1982b

The crop loss functions developed from the NCLAN programme appear to be the most promising method of developing quantitative estimates of oxidant effects to agricultural crops in Canada. The simple crop loss functions listed in Tables 4.3-20 and 4.3-21 would be useful as a first approximation for those crops in high ozone areas for which sufficient input data are available on a regional basis. These have the advantages of being easy, inexpensive and fast. NCLAN is now in the process of reviewing and calibrating existing crop growth and process models, so that a more effective predictive capability may be available in the next few years (Heck et al, 1982b).

Numerous studies have examined interactions between O_3 and other air pollutants, principally SO_2 (Flagler and Younger, 1982a,b; Hofstra and Ormrod, 1977; Foster et al., 1983; Garrett et al.,1982; and Larsen et al., 1983). Greater than additive negative effects have been noted for soybeans exposed to SO_2 and O_3 (Heggestad and Bennett, 1981) and for red kidney beans (Oshima, 1978). No interaction was noted by Foster (1980) for potato tuber yield.

The U.S. EPA (1982a) has summarized recent literature which reports greater than additive effects on yield on growth for SO_2 and O_3 in soybean; less than additive effects for cottonwood, yellow poplar, hybrid poplar, apple, marigold, and soybean; and strictly additive effects for petunia, begonia, radish and soybean.

Larsen et al. (1983) described an O_3 -SO_2 leaf injury mathematical model that expresses percent leaf injury as a function of simultaneous exposures to O_3 and SO_2 for soybean data. They conclude that the two pollutant model should be used when the ambient SO_2 concentration for an hour is about 5 times the O_3 level. When SO_2 concentrations are above the two-pollutant model limits, a one-pollutant SO_2 model should be used. Below the limits, a one-pollutant model for O_3 is used. Daily injury is determined by summing, by hour, impact calculated from the appropriate model.

4.3.4 Acid Precipitation

Direct Effects on Vegetation

Various types of injury may result from direct exposure of plants to acidic deposition (MOI, 1983; Cowling, 1979; Cowling and Dochinger, 1980; Tamm and Cowling, 1977). These include:

- damage to protective surface structure such as cuticle
- interference with normal functions of guard cells
- poisoning of plant cells, after diffusion of acidic substances through stomata or cuticle
- disturbance of normal metabolism or growth processes, without necrosis of plant cells
- alteration of leaf-and root-exudation processes
- interference with reproductive processes
- synergistic interaction with other environmental stress factors
- accelerated leaching of substances from foliar organs
- increased susceptibility to drought and other environmental stress factors
- alteration of symbiotic associations
- alteration of host-parasite interactions

Studies for the Memorandum of Intent (MOI, 1983) note that in contrast to results with O_3, experimental studies with simulated acidic deposition have produced both positive and negative results, since increases, decreases and no change situations have been reported. Results appear to depend on concentration of acids, plant species and cultivars, pattern and timing of rain applications, and soil, environmental and cultural conditions. The reviewers noted that each species may thus have unique patterns of physiological and genetic responses to the potentially beneficial and detrimental components of acidic deposition.

Evans et al. (1981) defined acidic injury to terrestrial vegetation as follows:

- loss of crop yield and/or quality
- visible injury which would reduce market value
- loss of forest yield or long-term growth of trees
- visible injury to ornamental plants that would reduce their aesthetic value
- substantial alterations of plant community composition leading to ecosystem simplification
- changes in herbivore populations or communities leading to any of the above
- altered sensitivity to other air pollutants and/or plant pathogens

Due to the variability in species response noted previously, it has been necessary to study effects on a species by species basis, so that an overall picture of which plant groups are sensitive and the types of effects possible is only now becoming apparent.

Evans (1982) concluded that there is little unequivocal evidence that acidic precipitation, at ambient levels, is having deleterious effects upon terrestrial vegetation in the United States, based upon a lack of concrete information rather than an evaluation of a large data base.

Loss of Crop Yield: Since managed soils are less subject to acidification than are natural systems due to fertilization and lime application (McFee, 1979) ill effects of acid precipitation on plants would likely result, if at all, from exposure to foliage (Evans et al., 1981). Simulated acid rain applied only to wet the foliage caused significant reductions in soybean yield and quality under experimental field conditions using standard agronomic practices (Evans et al., 1980).

Agricultural practices, in another sense, however, may substantially increase the potential for crop damage in the field from acidic deposition (MOI, 1983). Economic constraints in any given area and year tend to result in the exposure of extensive areas of a give crop in a relatively uniform state of plant development. The onset of the cycle of flowering physiology, pollen dispersal and fertilization, and photosynthetic partitioning, could all be potentially susceptible to damage over large areas.

Visible Injury and Reduced Market Value: If the foliage or fruits of some crops exhibit blemishes, market value is reduced. Simulated acidic rain has induced lesions on leaves and reproductive structures, up to a pH of 4.0 (Evans et al., 1981; Lee et al., 1980). No relationship between crop yield and foliar injury has been established, and there is no direct evidence that acidity levels of current rainfalls injure field or forest plant foliage (Evans et al., 1981). Field-grown plants may be less susceptible to the development of foliar symptoms than plants grown under controlled conditions (MOI, 1983; Jacobson, 1980) but the synergistic effects with O_3 and other pollutants are not yet known.

Loss of Forest Yield or Long-term Growth: Evans et al. (1981) noted conflicting reports as to whether acidic precipitation can influence forest productivity. Presumably, leaf injury in sufficient quantity might lead to a reduction in yield but this has not yet been ascertained. Benefits to trees may also occur through more rapid transfer of elements to the soil which provides opportunities for enhanced uptake and recycling. The amount of nitrate, and to a lesser extent sulphate and other elements, in acidic precipitation is unlikely to enhance agricultural yields, but may represent a significant nitrogen source to forests (Evans et al., 1981)

Foliar Injury to Ornamental Plants: Little research interest has been shown. Evans et al., (1981) noted that there is no evidence showing visible foliar injury to ornamental plants due to realistic precipitation acidities (excluding point source studies) that would reduce market or aesthetic value.

Alterations to Plant Communities: Evans et al. (1981) discussed the possibility that if one or more plant species were to be preferentially affected by acid precipitation so they become reduced or eliminated, then ecosystem simplification would result, eventually perhaps affecting overall ecosystem productivity. Ferns were cited by those authors as being sensitive, but the inhibition of ferns or other plants have not yet been related to ecosystem simplication.

Alterations in Herbivores: In the case that acidity may directly or indirectly alter populations of herbivores, change might be reflected in crop or forest productivity, but no data are currently available on such effects if they occur (Evans et al, 1981).

Altered Sensitivity of Vegetation: Epidermal cells may be injured upon exposure to rain at pH of 3.4 and below (Evans and Curry, 1979) perhaps altering gas exchange and therefore sensitivity to other air pollutants. Further specific work on combinations of ambient acidity levels on host-plant pathogen interactions are unknown, but if rainfall acidity were to change, the degree to which plant pathogens affect either crop on forest plants may have economic impacts (Ridgway et al., 1978; Shaw, 1979).

4.3.5 Summary of Quantifiable Effects

Nitrogen Oxides

There is little evidence to demonstrate that exposure to NO_x alone at low levels will lead to significant growth and yield reductions for commercially important crops and indigenous vegetation.

A predictive model (Heck and Tingey, 1979) is reported for foliar injury from acute NO_2 exposures. From this, Table 4.3-4 presents projected exposure concentrations and duration estimates required to produce foliar injury on susceptible, intermediate and tolerant plants at the threshold injury level. This information, in combination with specific NO_x concentration data and an inventory of sensitive vegetation types, could be used to assess injury to ornamental or garden plants in urban settings, as well as rural agricultural crops or natural ecosystems.

Photochemical Oxidants

Studies suggest that reponses to ozone are highly species dependant, and that a much more complex predictive modelling approach is required for damage assessment. Unlike NO_x, damage to natural ecosystems and crops is being experienced due to O_3.

There does not appear to be a functional methodology for assessing the effects of O_3 on forests or other natural ecosystems.

With regard to agricultural crops, we feel that yield is a better measure of adverse effects of O_3 than is foliar injury. Regression models for selected crops have been developed and tested to an extent as part of the National Crop Loss Assessment Network (NCLAN) in the United States that appear to be useable, with verification, in Canadian settings where O_3 data and some yield loss data are available. These were reported by Heck et al. (1982a, b) and are summarized in Tables 4.3-16 and 4.3-17 for foliar injury and crop yields/losses respectively.

Information in Tables 4.3-19 to 4.3-21 provides pollutant dose/crop response functions, percent yield reductions as a function of O_3 concentration, and crop loss functions for major species which provide a basis for evaluating oxidant effects on several prominant crops.

Reviews by Ormrod et al. (1980) and Pearson (1983) suggest that crop injury in Ontario due to oxidants was of prime concern for the following crops: white bean, tobacco, potato and tomato, with injuries to grape leaves and onions also being cited.

Unfortunately, for the most part, these crops have not been subjected to rigorous examination in the NCLAN programme in the United States. As reported in Tables 4.3-20 and 4.3-21, crop loss functions have been developed for soybean, winter wheat, corn and peanut. Additional yield reductions have been examined for kidney bean, head lettuce, spinach, turnip and wheat. While many of these are important crops in parts of Canada, they do not coincide well with the Ontario data base for oxidant-related crop damage.

We recommend, for the purposes of this SEIA study, that the "all other" crop loss function, developed by Heck et al. (1982b) and reported in Table 4.3-21, be used as a first approximation of change of crop yield as a function of O_3 dose. The linear model is as follows:

$$Y = 13.0 + 519x$$

where Y = yield as % reduction

x = seasonal 7h/day mean O_3 concentration (ppm)

and is intended as a broad guide in estimating crop losses for all species not specifically listed. It is intended for use with broad groups of crops and should not be used for estimating losses to any single species (eg. radish or carrot) (Heck et al. 1982b). As a first rough approximation, derived on a regional basis where the ambient O_3 data base is sufficient, this approach appears to be the most valid and cost effective approach to estimating crop losses due to ozone.

In portions of the country where wheat and corn are widely planted, the specific crop loss functions for those species (Table 4.3-21) can be utilized, at least on a tentative basis. We note that the background research used to derive these models was undertaken in the United States, and such factors as climate, soils and the genetic make-up of the Canadian vs U.S. crops should be examined prior to estimation of yield loss.

The O_3/SO_2 leaf injury model reported by Larsen et al. (1983) was developed using data for soybean, but interactions among these pollutants have shown wide variation in the literature, even for soybean so that use of this model in Canada is perhaps premature. In any event, it uses foliar injury as a measure of effect rather than yield reduction.

Acid Precipitation

No documented losses of agricultural crops, forests, or horticultural plants appear to have been clearly identified with acid precipitation in North America. No methods exist which either identify or quantify adverse effects on terrestrial ecosystems, although we have listed a number of potential effects in the text.

4.4 Aquatic Environment

4.4.1 Introduction

This section examines the effects of automobile emissions on the aquatic environment. Of the emission components being examined at this time, only nitrogen oxides (NO_x) appear to have any potential for widespread or significant adverse effects on aquatic systems. Through atmospheric transformations of NO_x to nitrate (NO_3^-) and the formation of nitric acid (HNO_3) in wet deposition, these emissions are contributors to the phenomenon known as acidic precipitation. In the section which follows, we will describe the known effects of acidic precipitation on the aquatic environment, and discuss the relative importance of NO_3^- and HNO_3 to the direct and indirect acidification of Canadian freshwaters. The role of airborne nitrogen compounds as nutrients in aquatic ecosystems will also be considered.

Acidic precipitation has a pH less than 5.0-5.6; the upper pH value is the average value of uncontaminated precipitation in equilibrium with ambient carbon dioxide (see Chapter 2). Harvey et al. (1981) reported that in most cases, the parameter of interest is the deposition of acidic and acidifying substances rather than precipitation pH. Acidic substances include strong acids (e.g. sulphuric, nitric and hydrochloric acids), whereas acidifying substances are protolytes which can act as acids under certain conditions.

Substances transported through the atmosphere come to earth as wet deposition (precipitation) and dry deposition. Current knowledge suggests that wet and dry deposition are of approximately equal importance on a regional basis in Canada (Harvey et al., 1981). The low pH is explained by the presence of the strong acids H_2SO_4 and HNO_3, with weak acids probably being relatively unimportant (Galloway et al., 1976). The relative abundance of these two acids varies considerably across Canada (MOI, 1983), as do concentrations of the four ions usually of most importance to rainfall acidity: hydrogen (H^+), ammonium (NH_4^+), nitrate (NO_3^-) and sulphate (SO_4^{2-}). A portion of the nitrogen and sulphur-containing pollutants is oxidized to nitric and sulphuric acids, so that the acid content of precipitation can be considered a secondary result of the primary anthropogenic emissions

(Whelpdale, 1980; Logan, 1982).

4.4.2 Water Quality

Of the ions which largely account for acidic deposition, most studies now suggest that based on documented effects, wet and dry deposition of sulphur compounds dominate in long-term acidification (MOI, 1983). As well, Agnew et al. (1982) have reported there is strong evidence to show that SO_2^{2-} dry deposition is not only the most important contributor to snowpack pollutant mass, but also the major contributor to snowpack acidity. Thus, sulphur compounds appear to be of importance in both long-term and short-term acidification of surface waters.

The primary nitrogen compounds in wet deposition are NO_3^- and NH_4^+. Of the two, only NO_3^- is associated with the acidity of wet deposition (Galloway and Dillon, 1982). Dry deposition of nitrogen oxides appear to be of equivalent magnitude to wet deposition of these compounds (Logan, 1982; Galloway and Dillon, 1982).

In terms of acidification of receiving waters, concerns relate to pollutants which contribute H^+ to the systems or which cause the release of hydrogen ions once in the systems (Harvey et al., 1981). These include sulphur and nitrogen oxides and ammonium ion.

The transformations and fate of nitrogen compounds affecting the acidification of surface waters have been summarized by McLean (1981) and Galloway and Dillon (1982). Much of the atmospheric N falls within the terrestrial portion of the watershed, so that biological and geological processes affect water quality prior to reaching lakes or streams.

As noted earlier, the deposition of NH_4^+, NO_x, and NO_3^- affects the acid-base balance of the receiving system. Galloway and Dillon (1982) noted that since the dry deposited NO_x is probably rapidly oxidized to HNO_3, it is reasonable to limit the discussion of the effect of atmospheric N on terrestrial ecosystems to only NH_4^+ and NO_3^-. Those authors determined that once in the terrestrial ecosystem, NH_4^+ has three potential fates:

- NH_4^+ may be oxidized to NO_3^-, producing two equivalents of acidity for every equivalent of NH_4^+ oxidized
- it may be taken up by a plant, producing one equivalent of acidity for each equivalent of NH_4^+ taken up
- NH_4 may be discharged from the soil solution into the lake

Galloway and Dillon (1982) noted that the last of these possibilities is least important since watersheds retain most NH_4^+ deposited, so that atmospheric deposition of ammonium always has an acidifying effect on the terrestrial system.

Similarly, Galloway and Dillon (1982) examined the fate of deposited NO_3^- in terrestrial systems and found it may be retained through:

- assimilatory (uptake) reduction which will produce a maximum of one equivalent of alkalinity for each equivalent of NO_3^- assimilated by the plant
- dissimilatory (denitrification, ammonification) reduction which will produce a maximum of two equivalents of alkalinity for each equivalent of NO_3^- reduced
- discharge from the soils solution to the surface waters

The authors noted that the first process mentioned consumes strong acids and releases OH^-, but that the amount consumed may be less than the amount of NO_3^- taken up (Raven and Smith, 1976), so that the nitrate assimilated and the alkalinity generated may only in part counter the strong acids associated with the nitrate.

Both McLean (1981) and Galloway and Dillon (1982) point out that in the reduction reactions of nitric acid, H^+ is used up, with the amount depending on other conditions, but in general, the more nitrate that is utilized, the more acid is consumed. If the terrestrial system were a perfect sink and NH_4^+ deposition were greater than or equal to NO_3^- deposition, the system would become acidified. If, on the other hand, NO_3^- deposition were greater, acidification by NH_4^+ may be partially or totally offset by the NO_3^- and, in fact, net alkalization may even occur.

Since in most of eastern North America, more nitrate is deposited than ammonium (Scheider et al., 1979), the impact of the NH_4^+ may be largely offset by the NO_3^- if the NO_3^- is retained in the terrestrial ecosystem (Galloway and Dillon, 1982). If the cation accompanying the deposited NO_3^- is H^+, then this alkalization of the terrestrial system would be lessened. It should be noted that alkalinity production depends upon uptake of NO_3^- by plants and/or reduction to NH_4^+.

Galloway and Dillon (1982) noted that terrestrial systems are not sinks for all the NH_4^+ and NO_3^-, and during winter and spring snowmelt, large amounts of NO_3^- are released from the terrestrial system to the surface waters. If the accompanying cation is H^+, then acidification of the aquatic system will result, and if a base cation, then a loss of nutrients from the terrestrial system will occur.

Since soils and plants are not passive reservoirs through which the N passes (Galloway and Dillon, 1982; MOI, 1983), the longer the acidic deposition stays in contact with soil, the smaller the possibility of the N being transported to a lake, and therefore, there is a reduced chance of a direct effect on the aquatic system due to the atmospheric deposition of N. In general, only during periods of high flow, such as spring melt or large rainstorms, or during winter, would a significant portion of the deposited N falling in the terrestrial portion of the watershed reach a lake or stream.

It should be noted that current evidence suggests that NO_3^- generally has a net alkalizing effect on terrestrial and aquatic systems, while NH_4^+ has a potential acidifying effect.

The sensitivity of surface waters to acidification relates not only to the above biological factors, but also to geochemical and hydrological characteristics of watersheds. Natural weathering processes consume H^+ so that, in a sense, the acidification process may be viewed as an acceleration of this weathering process in that H^+ concentrations are greater, reaction rates are faster, and surface waters may receive elevated inputs of H^+, cations, and other substances (Harvey et al., 1981). Position in the watershed and relative proportions of surface to ground water inputs may affect the acid neutralizing capabilities of lakes in similar geographic locations.

The degree of sensitivity of aquatic ecosystems to acidification by external loading of H^+ can be determined by the base saturation component of the total exchange capacity and by direct buffering capacity within the hydrologic system associated with lakes and streams. Harvey et al.(1981) and MOI (1983) described sensitivity mapping of Canadian surface waters based on bedrock and soil characteristics prepared by RCG (1979), Elder (1980) and Shilts (1980). Additional sensitivity mapping for eastern Canada based upon this work, as well as Lucas and Cowell (1982) and Olsen et al. (1982) for the eastern portions of Canada and the United States, were prepared by Impact Assessment Work Group I for the United States-Canada Memorandum of Intent on Transboundary Air Pollution (MOI, 1983). Complete agreement on the validity of this sensitivity was not reported by Canadian and American contributors to that study at the time of writing.

Based upon the major ion chemistry of surface waters, many areas in Quebec and the Atlantic Provinces are sensitive to acidic deposition (Harvey et al., 1981), while for much of Ontario, the glacial overburden is heterogeneous, with the result that significant variation in sensitivity occurs. Those authors also noted that numerous areas in eastern Canada that are classified as sensitive are also located in areas of elevated acidic deposition.

With respect to N deposition, Galloway and Dillon (1982) concluded that as long as the terrestrial system acts as a sink for atmospheric nitrogen compounds and therefore prohibits their release to the aquatic system, there will be no intermediate or long-term effects of nitric acid on aquatic systems. The authors noted that if the terrestrial system were to become saturated with nitrogen, then nitrate would begin to be released to the surface waters along with an equivalent amount of cations. If the cation is H^+, then acidification would result, whereas if it is Ca^{++}, Mg^{++} etc, then the soil will lose nutrients. At this time, Galloway and Dillon (1982) reported that it is unknown if forests will become saturated with respect to nitrogen, or how long this process would take.

In terms of short-term effects of N deposition, McLean (1981) reviewed North American and Scandanavian studies and found that nitric acid tends to accumulate in the snowpack over the winter, and during rapid melt runoff, could have much higher than normal amounts of nitric acid, during a time of particular sensitivity to the life cycles of many aquatic species. McLean's assessment was that nitric acid contributes slightly less than sulphuric acid to the observed drops in stream and lake pH following snowmelt.

Galloway et al. (1980, 1982) found that in their study of lakes, the alkalinity decreased during snowmelt because of dilution of base cations in the lake epilimnion by snowmelt water and an increase in NO_3^- in the epilimnion, and concluded that the source of NO_3^- was the snowpack. Galloway and Dillon (1982) noted that although SO_4^{2-} concentrations changed only slightly during snowmelt, SO_4^{2-} still contributes to the acidification in an indirect manner by causing long-term as opposed to episodic alkalinity reductions. Thus, the short-term reduction of alkalinity due to NO_3^- is added to the long-term reduction in alkalinity due to SO_4^{2-}. As a general rule of thumb, Galloway and Dillon (1982) reported that in lakes with alkalinities near 0 µeq/L, increases in NO_3^- are the most important reason for acidification during snowmelt. Since the hydrological pathway that the snowmelt follows, and the volume of the lake that interacts with the runoff have rarely been investigated, considerable effort would be needed in these and other areas before a model could be constructed and verified which would predict the impact of these episodic events on aquatic ecosystems by determining the probability of chemical change of specific magnitude in a lake or stream for a specified duration.

In addition to the direct effects of acidification and N deposition discussed above, other aspects relating to water quality require some consideration. Nitrogen is a major plant nutrient, the lack of which limits growth in nature particularly in terrestrial ecosystems. Evans et al. (1981) reported that inputs of N in polluted rain, while they do not fulfill forest N requirements, do appear to make a significant positive contribution as a fertilizer although N inputs of this magnitude were thought to be insignificant relative to crop needs. NO_3^- inputs to freshwaters also are utilized as nutrients by aquatic vegetation, but no quantitative analysis of the importance of N deposition to aquatic productivity appears to have been reported in the literature. However, uptake of nitrate ions by algae and aquatic plants results in the production of alkalinity in surface waters (Goldman and Brewer, 1980; MOI, 1983).

The U.S EPA (1978) has reported that phosphorus and nitrogen are the most important nutrients that stimulate eutrophication, and in most lakes, phosphorus is considered the more critical of the two. In coastal and estuarine ecosystems, however, nitrogen is often the limiting nutrient and inputs of this element may control eutrophication. The authors noted that in many already-eutrophic lakes, biotic productivity is controlled by nitrogen, because the N/P ratios of pollutants from many cultural sources (e.g. domestic sewage) are far below the ratios needed for plant growth.

The average atmospheric input of 10 to 20 kg/ha-yr that is typical for most of the United States was thought to be a sufficient nutrient loading to support a moderate increase in biotic productivity in some lakes, especially shallow, oligotrophic lakes that may be nitrogen-limited (EPA, 1978). Those authors felt that atmospheric nitrogen fluxes may contribute to slight eutrophication in such cases, but that it is unlikely that these inputs alone would induce serious water quality problems.

Andren et al. (1977) summarized atmospheric loadings to the Great Lakes, and reported the following values for nitrogen (g/ha/yr):

Michigan	Superior	Huron	Erie	Ontario
no data	6,800	8,700	13,600	10,800

The authors noted that the dominance of bulk sampler data resulted in a conservative estimate due to loss of NH_3 from the samples before analysis. The distribution of deposition rates indicates a strong north to south gradient which was believed to relate to emissions from transportation and other combustion sources and from the more intense agricultural activities.

Bennett (1982) noted that most of the nitrogen input to Lake Superior originates outside of the lake drainage basin and is by way of atmospheric deposition directly onto the lake surface. The calculated trend in loading parallels that of emissions of anthropogenic nitrogen oxides in the industrial Midwest and Great Lakes areas of North America, although evidence is circumstantial. The author noted that Lake Superior is outside the zone of highest nitrogen emissions, but that loadings still amount to about 40 percent of the highest rates, which are in the Lake Erie region. The 1973 nitrogen budget for Lake Superior showed that of the 95,000 tonnes total input, 56,000 tonnes (60%) was due to precipitation directly on the lake surface. Of the 86,000 tonnes falling in the terrestrial portion of the watershed, much was utilized by terrestrial vegetation, but 36,000 tonnes still reached the lake via tributaries.

According to Bennett (1982) it is clear that atmospheric loading provides most of the nitrogen input to Lake Superior and that the trend of nitrogen concentration in the lake should reflect long term trends in atmospheric loading. He noted that eutrophication is an unlikely problem for Lake Superior because its present oligotrophic state is due to a low concentration of total phosphorus, independent of the nitrogen intent.

Another aquatic effect relates to changes in dissolution of soil and bedrock components in acidic conditions. Watersheds containing carbonate minerals (e.g. calcite, dolomite) appear to have an almost unlimited capacity for buffering H^+ input, with cations such as Ca^{2+} and Mg^{2+} being exported at an increased rate (Harvey et al., 1981). However, in watersheds dominated by aluminosilicate minerals, adsorption and dissolution reactions at lower pH values may result in increased concentrations of aluminum and other metal species which may interact with aquatic organisms and nutrient cycling leading to indirect impacts. Similarly, some concerns have been raised over acidification liberating increased amounts of mercury and other metals which may build up in the food chain or affect drinking water supplies (see Section 4.2, Health Effects).

4.4.3 Fish and Aquatic Organisms

Numerous workers have examined aspects of the effects of acidic precipitation on aquatic biota within the last decade. To a great extent, these investigations have centred in Scandinavia, Canada and the United States, where impacts were first recognized and found to be of major importance. Recent reviews by MOI (1983), Harvey et al. (1981), Evans et al. (1981), Flett (1981), Haines (1981) and Beamish (1976) summarize the known impact data base. Wiener (1983) has prepared a recent selected annotated listing of information sources related to acidic precipitation. The discussions which follow are derived principally from these reviews.

Microorganisms

Harvey et al. (1981) noted that very little information is available on the role of microorganisms in ecosystems affected by acidic deposition. Sulphur cycle bacteria may in some circumstances contribute to the neutralizing capacity of surface waters by sulphate reduction. The authors also noted that some studies indicated that microbial decomposition of organic matter is markedly reduced at pH levels encountered in acidic lakes. Harvey et al. (1981) noted that it is believed that such disruptions of detrital trophic structure would affect nutrient cycling and hence other trophic levels.

In a laboratory study, Bick and Drews (1973) demonstrated that as pH was lowered, the number of protozoans and bacteria decreased, populations of fungi increased, and the rates of decomposition and nitrification were reduced. Rao et al. (1982) observed reduced numbers of nitrifying bacteria and sulphur cycle bacteria in low pH lakes and streams.

Planktonic Communities

The biomass of phytoplankton in acidic lakes is not significantly different from non-acidic lakes with similar phosphorus concentrations (Harvey _et_ _al_., 1981), and productivity is not reduced although the distribution of biomass among the algal classes differs. The authors noted that whereas chrysophytes or diatoms normally constitute most of the biomass in oligotrophic Shield lakes, these classes are replaced by dinoflagellates under acidic conditions. Within each class of phytoplankton, the number of species is found to decrease with declining pH in Shield lakes. Species diversity was markedly reduced in those LaCloche Mountain lakes (central Ontario) having pH's below 5.6 (Kwiatkowski and Roff, 1976). Nicholls _et_ _al_. (1981) found that foul odours developed in acidic recreational lakes, apparently due to the growth of the planktonic _Chrysochromulina_ _breviturrita_.

Acidification apparently results in reduced zooplankton biomasses, as both the numbers and average size of community numbers are reduced (Yan and Strus, 1980; MOI, 1983). As a result, food availability to higher trophic levels may be decreased. Harvey _et_ _al_. (1981) reported that in lakes having pH's below 5.0, the occurrence and relative abundance of several crustacean species were reduced, such as species of cladocerans and cyclopoid copepods. The dominance of _Bosmina_ _longirostris_ and _Diaptomus_ _minutis_ becomes even more evident in oligotrophic Shield lakes which are acidic, perhaps related to a reduction in invertebrate predation. Harvey _et_ _al_. (1981) noted that reduced abundance and diversity of planktonic rotifers have been observed in Swedish and Canadian lakes. Little information is available on the effects of acidification on aquatic insects or protozoans.

While it is often assumed that the direct cause of changes in the zooplankton community is related to differences in tolerance among species to increased H^+ concentrations, other factors may contribute. Acidification increases the transparency of lakes, increases the concentration of potential toxicants, and produces quantitative and qualitative changes in zooplankton predator and prey species (MOI, 1983; Harvey _et_ _al_., 1981). Therefore, the actual causes of observed changes in community structure may be quite complex.

Benthic Communities

Harvey et al. (1981) noted that with the exception of some Swedish field studies, there is little information available on the effects of acidification on benthic macrophytes. Sphagnum has been identified in European acidified lakes, but this has not been a major phenomenon in Ontario Shield lakes. Sphagnum coverage of littoral areas creates a unique habitat that is considered unsuitable for other macrophytes and many invertebrates (MOI, 1983). Harvey et al. (1981) noted that acidic Ontario Shield lakes had diversified plant communities which included most of the rosulate and isoetid species common to Canadian Shield lakes. Acidified lakes and streams are often characterized by increased growth of filamentous algae which may compete with other plant forms and cause some depreciation of shoreline recreational values and activities, particularly swimming (MOI, 1983).

Aquatic invertebrate species are known to be affected by low pH conditions (MOI, 1983) with molluscs being highly sensitive (require >pH 5.8 to 6.0) and oligochaetes being found in reduced densities in acidic waters (Wiederholm and Eriksson, 1977). Orders of insects have varying sensitivities, with the number of species of Ephemeroptera and Plecoptera appearing to be positively correlated with pH, while larvae of Chironomidae (Diptera), Hemiptera and Megaloptera are often abundant in acid lakes (MOI, 1983; Almer et al., 1978).

No casual relationship between H^+ concentration and effects on invertebrate communities has yet been identified (MOI, 1983). Other factors varying with pH include nutrients, bicarbonate and several metals, and it appears that molluscs and moulting crustaceans, with their sizeable calcium requirements, may be the macroinvertebrates most sensitive to low pH. The relationship between the benthos and benthic feeding fish in acidifying lakes is not known (Harvey et al., 1981).

Fish Communities

Harvey et al. (1981) have provided the most detailed summary of the affects of acidification on Canadian fish populations. The authors noted that affected areas of Canada include the LaCloche Mountains, the Sudbury region, the Wawa area, south-central Ontario and eastern Nova Scotia. Lakes exist from which all fish have been lost, while from others, individual species populations have disappeared. Those surviving in acidifying waters are reduced in abundance, altered in age composition, or are under physiological stress. No systematic tally exists of lakes with lost or acid stressed fish populations.

Beamish (1976) identified the approximate pH at which fish in the LaCloche Mountain lakes stopped reproducing (Table 4.4-1). Prized game species such as smallmouth bass, and walleye (pickerel) had reproductive failure between pH 5.5 and 6.0, suggesting low tolerance of acidifying conditions. More tolerant species, even to pH 4.5 to 4.7, included lake herring, yellow perch, and lake chub.

An estimated 140 Ontario lakes, many located in the Sudbury area, have lost all fish populations (OME, 1979), while seven former salmon rivers in Nova Scotia are unsuitable for successful reproduction of Atlantic salmon due to low pH (Farmer et al.,1980), while additional waters were identified as being threatened. Additional losses have been documented in the United States (Evans et al., 1981), Norway and Sweden.

In acidifying lakes which still contain fishes, the populations may be reduced in abundance (Harvey et al., 1981; Harvey, 1979). Fish populations close to their pH tolerance limit are usually very small and probably are in their last stage prior to extinction. Figure 4.4-1 summarizes frequency of occurrence of fish species in relation to pH for LaCloche area lakes, identifying unaffected, stressed, and extinction levels.

TABLE 4.4.-1

—Species of fish that ceased reproducing, declined, or disappeared from natural populations as a result of acidification from acidic precipitation, and the apparent pH at which this disappearance occurred.

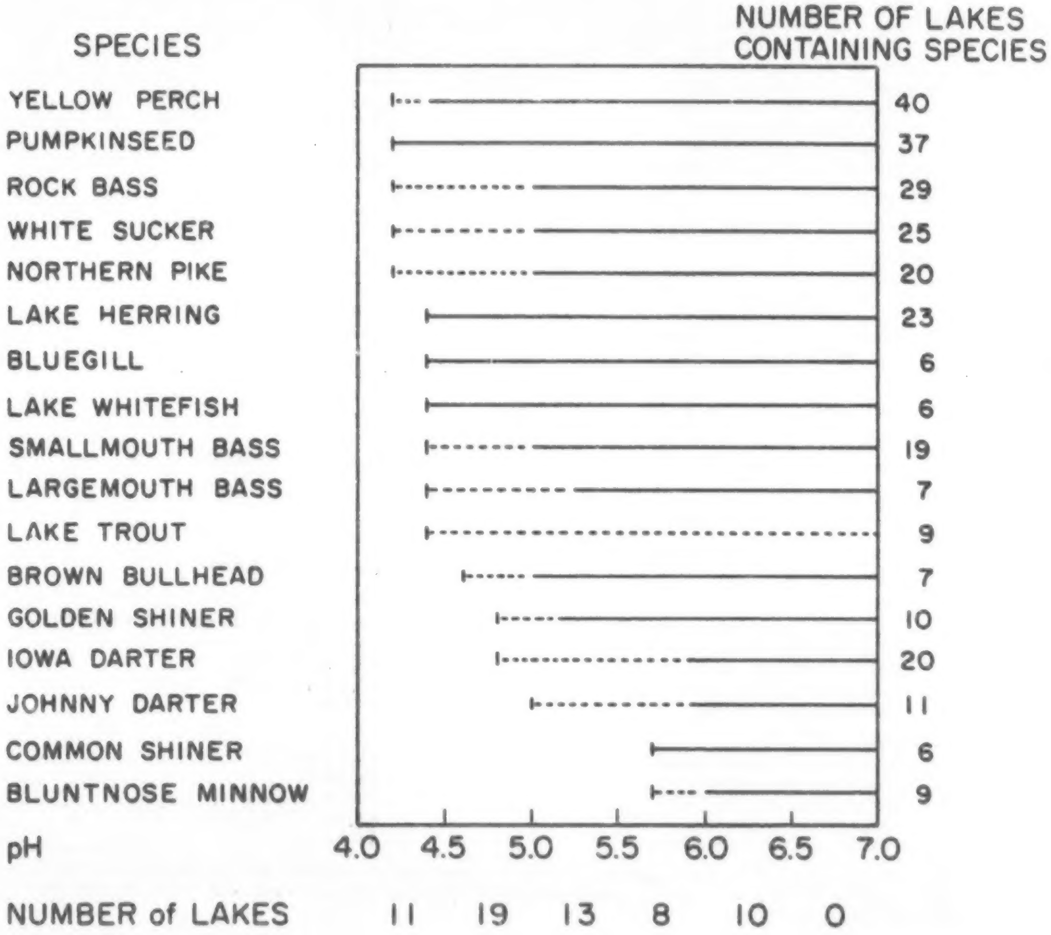
Family and species	Apparent pH at which population ceased reproduction, declined, or disappeared (reference)[a]
Salmonidae	
Lake trout *Salvelinus namaycush*	5.2–5.5 (1); 5.2–5.8 (2); 4.4–6.8 (3)
Brook trout *Salvelinus fontinalis*	4.5–4.8 (4); ~5 (5)
Aurora trout *Salvelinus fontinalis timagamiensis*	5.0–5.5 (6)
Arctic char *Salvelinus alpinus*	~5 (7)
Rainbow trout *Salmo gairdneri*	5.5–6.0 (4)
Brown trout *Salmo trutta*	5.0 (4); 5.0–5.5 (8); 4.5–5.5 (9)
Atlantic salmon *Salmo salar*	5.0–5.5 (4)
Lake herring *Coregonus artedii*	4.5–4.7 (1); <4.7 (2); 4.4 (3)
Lake whitefish *Coregonus clupeaformis*	<4.4 (3)
Esocidae	
Northern pike *Esox lucius*	4.7–5.2 (2); 4.2–5.0 (3)
Cyprinidae	
Golden shiner *Notemigonus crysoleucas*	4.8–5.2 (3)
Common shiner *Notropis cornutus*	<5.7 (3)
Lake chub *Couesius plumbeus*	4.5–4.7 (1)
Bluntnose minnow *Pimephales notatus*	5.7–6.0 (3)
Roach *Rutilus rutilus*	5.3–5.7 (7)
Catostomidae	
White sucker *Catostomus commersoni*	4.7–5.2 (1,2); 4.2–5.0 (3)
Ictaluridae	
Brown bullhead *Ictalurus nebulosus*	4.5–5.2 (1,2); 4.6–5.0 (3)
Percopsidae	
Trout-perch *Percopsis omiscomaycus*	5.2–5.5 (1)
Gadidae	
Burbot *Lota lota*	5.5–6.0 (1); 5.2–5.8 (2)
Centrarchidae	
Smallmouth bass *Micropterus dolomieui*	5.5–6.0 (1); >5.5 (2); ~5.8 (10); 4.4–5.0 (3)
Largemouth bass *Micropterus salmoides*	4.4–5.2 (3)
Rock bass *Ambloplites rupestris*	4.7–5.2 (1,2); 4.2–5.0 (3)
Pumpkinseed *Lepomis gibbosus*	4.7–5.2 (1); <4.2 (3)
Bluegill *Lepomis macrochirus*	<4.2 (3)
Percidae	
Johnny darter *Etheostoma nigrum*	5.0–5.9 (3)
Iowa darter *Etheostoma exile*	4.8–5.9 (3)
Walleye *Stizostedion v. vitreum*	5.5–6.0 (1); 5.2–5.8 (2)
Yellow perch *Perca flavescens*	4.5–4.8 (1); <4.7 (2); 4.2–4.4 (3)
European perch *Perca fluviatilis*	5.0–5.5 (11)

[a] References: (1) Beamish 1976; (2) Beamish et al. 1975; (3) Harvey 1980; (4) Grande et al. 1978; (5) Schofield 1976; (6) Anonymous 1978; (7) Almer et al. 1974; (8) Jensen and Snekvik 1972; (9) Wright and Snekvik 1978; (10) Pfeiffer and Festa 1980; (11) Runn et al. 1977.

Source: Haines (1981)

Figure 4.4-1

Frequency of occurrence of fish species in six or more La Cloche Mountain lakes in relation to pH. Vertical bar, lowest pH recorded; dashed line, stressed populations; solid line, populations which appear unaffected (from Harvey 1979).



The mechanisms of fish injury are still being clarified, but the most obvious one is the failure of recruitment of new age classes into the population. Several studies have shown failure to spawn, impaired hatching, and larval sensitivity at the critical stages of early life history where lower pH may have severe negative impacts (Harvey et al., 1981; Evans et al., 1981). Osmotic regulatory difficulties seem to be responsible for many observed effects with altered Na^+, Cl^-, and K^+ levels being observed in plasma and muscle tissues.

Increased metal levels appear to be implicated in fish mortality, particularly in the case of concentrations of toxic aluminum species related to increased solubility at reduced pH. Elevated levels of aluminum in waters have been shown to have serious effects on fish within the pH range normally considered not harmful to aquatic biota (Baker and Schofield, 1980). The aquatic chemistry of aqueous aluminum has been described by Spry et al. (1981). Toxic aluminum concentrations have been associated with disturbed ion exchange over gills and with respiratory distress. Low pH has also been associated with the formation of methyl mercury, a stable and soluble form which readily bioaccumulates, but evidence suggesting mercury toxicity being a widespread cause of fish stress is lacking. The public health implications of increased mercury concentrations in food species of fish due to increased methylation at low pH are now receiving some consideration (Harvey et al.,1981; Scheider et al., 1979).

Effects on altered fish food supply, especially in the pH range of 5.0 to 6.0, on fish condition or community composition have not been studied (Evans et al., 1981), even though lake acidification may cause drastic alterations of plant and invertebrate communities in lakes. The authors noted that, although there is little doubt that physiological effects of H^+ and Al are the principal causes of complete elimination of fish from most acidic waters, slight effort has gone into investigating the consequence of changing food quantity or quality at intermediate acidity levels (pH-5.0 to 6.0), which may be a critical factor to survival and growth of newly hatched fish.

Other Semi-aquatic Organisms

Harvey et al. (1981) noted that the importance of amphibians in aquatic and terrestrial ecosystems tends to be ignored, although recent work has done much to clarify the sensitivity of this group of herptiles. Clark and Fischer (1981) ranked susceptibility of breeding habitat to pH depression for amphibians in northeastern North America whose ranges overlap areas receiving acidic deposition (Table 4.4-2). Nine species were classified as highly susceptible due to their use of acidic meltwater pools as breeding habitat (Harvey et al., 1981). Another dozen species were categorized as having moderate potential for acidification of egg laying habitat.

Certain mammals and birds may be subject to effects of lake acidification, primarily due to alteration of habitat conditions, or disruption of their food supply, either through species changes or productivity decline. Table 4.4-3 summarizes findings from MOI (1983) regarding potential impacts of this nature.

4.4.4 Relative Importance of Sulphur and Nitrogen Compounds to Acidification

Only recently have the relative roles of sulphur and nitrogen compounds been examined. Section 4.4.2 described how four major ions (H^+, NH_4^+, NO_3^-, and SO_4^{2-}) have some potential for altering surface water acidity. Studies by Galloway and Dillon (1982) and McLean (1981) suggest that interaction of nitrate ions with soils and vegetation allows nitric acid to be largely assimilated by the terrestrial portion of the watershed (MOI, 1983). Similarly, nitrate falling directly on a lake would be easily assimilated by aquatic vegetation under most conditions. Since there are few comparable terrestrial sinks for sulphur, SO_4^{2-} is a conservative ion whose export to surface waters is directly related to deposition in precipitation.

Impact Assessment Work Group 1 of the United States-Canada Memorandum of Intent on Transboundary Air Pollution (MOI, 1983) has listed additional aspects to the issue of the dominant anion associated with the acidification of surface waters, including:

TABLE 4.4-2

SUSCEPTIBILITY OF BREEDING HABITAT TO pH DEPRESSION FOR THOSE AMPHIBIANS IN NORTHEASTERN NORTH AMERICA WHOSE RANGE OVERLAPS AREAS RECEIVING ACIDIC DEPOSITION (modified from Clark and Fischer 1981)

Potential for acidification of egg-laying habitat	Habitat	Species
high	meltwater pools	Ambystoma maculatum - Yellow-spotted salamander
		Ambystoma laterale - Blue-spotted salamander
		Ambystoma tremblayi - Tremblays salamander
		Bufo americanus - American toad
		Pseudacris triseriata - Chorus frog
		Rana sylvatica - Wood frog
		Rana pipiens - Northern leopard frog
		Hyla crucifer - Northern spring peeper
		Hyla versicolor - Gray tree frog
moderate	permanent ponds	Necturus maculosus - Mudpuppy
		Notophthalmus viridescens - Red-spotted newt
		Bufo americanus - American toad
		Hyla versicolor - Gray tree frog
		Pseudacris triseriata - Chorus frog
		Rana catesbeiana - Bullfrog
		Rana clamitans - Green frog
		Rana pipiens - Northern leopard frog
		Rana septentrionalis - Mink frog
	streams	Eurycea bislineata - Northern two-lined salamander
		Necturus maculosus - Mudpuppy
	lakes	Rana catesbeiana - Bullfrog
low	bogs	Hemidactylium scutatum - Four-toed salamander
	logs and stumps	Plethedon cinereus - Red-backed salamander

Source: MOI (1983)

TABLE 4.4-3

AVIAN AND MAMMALIAN SPECIES MOST LIKELY TO BE INFLUENCED BY A REDUCTION IN FOOD RESOURCES DUE TO ACIDIC DEPOSITION, FEEDING HABITAT DURING THE BREEDING SEASON AND ALTERNATE FOOD RESOURCES

Susceptible Food Resources	Feeding Habitat During the Breeding Season	Species	Alternate Food Resources
Fish, aquatic invertebrates amphibians	Lakes, Rivers	Common Loon (*Gavia imuner*)	none
		Osprey (*Pandion haliaetus*)	none
	Littoral zone	Great Blue Heron (*Ardea herodius*)	none
		Belted Kingfisher (*Megaceryle alcyon*)	some terrestrial invertebrates
		Hooded Merganser (*Lophodytes culcullatus*)	some aquatic plants
		Ring-necked Duck (*Aythya collaris*)	some aquatic plants
		Common Merganser (*Mergus merganser*)	none
		American Mink (*Mustela vison*)	small mammals, birds
		River Otter (*Lontra canadensis*)	small mammals
Aquatic invertebrates	Littoral zone	Common Goldeneye (*Bucephala changula*)	fish
		Red-breasted Merganser (*Mergus serrator*)	fish
		Black Duck (*Anas rubripes*)	aquatic plants
		Green-winged Teal (*Anas carolinensis*)	aquatic plants
		Mallard (*Anas platyrhynchos*)	aquatic plants
		Northern Pintail (*Anas acuta*)	aquatic plants
		American Wigeon (*Anas americana*)	aquatic plants
	Riparian zone	Spotted Sandpiper (*Actitis macularia*)	none
Aquatic plants	Wetlands	Muskrat (*Ondratra zibethicus*)	aquatic invertebrates
		Common Shrew (*Microsorex hoyi*)	
Aquatic invertebrates	Wetlands and Riparian Zone	Common Yellowthroat (*Geothylpis trichas*)	some terrestrial invertebrates
		Bank Swallow (*Riparia riparia*)	
		Myrtle Warbler (*Dendroica coronata*)	
		Eastern Kingbird (*Tyrannus tyrannus*)	
		Blackpoll Warbler (*Pendroica straita*)	
		Northern Waterthrush (*Seiurus boracensis*)	
		Alder Flycatcher (*Empidonax alnorum*)	
		Wilson's Warbler (*Wilsonia pusilla*)	
		Lincoln's Sparrow (*Melospiza lincolnii*)	

Source: MOI (1983)

- the relative magnitude of SO_4^{2-} and NO_3^- in the rain and snow inputs, their variation during the year, and long-term trends
- the relative magnitude of the biological interactions of both anions in watersheds, as they are affected by biological activity at different seasons or by changes in biomass over long periods
- the production of alkalinity in terrestrial and aquatic systems when NO_3 is assimilated by plants
- the contact time of precipitation inputs with the watershed

Data examined (MOI, 1983) indicated that acidic sulphur inputs exceed acidic nitrogen inputs over eastern North America on an annual basis and the net yield of these anions to streams and lakes is predominantly SO_4^{2-} on an annual basis (Harvey _et_ _al_., 1981). The authors concluded that since nitrate reaches surface waters in small amounts relative to its loadings on an annual basis and does not accumulate in surface waters, its influence on long-term surface water acidification is less than sulphate.

Studies by the Ontario Ministry of the Environment (cited in MOI, 1983) show that as acidity increases, the relative importance of NO_3^- declines, and the acid concentration can be explained by the presence of sulphuric acid.

McLean (1981) and Galloway and Dillon (1982) have noted that nitric acid may play a more significant role in short-term acidification associated with spring melt of nitrate-containing snowpack. Thus, nitrate might be important on an episodic basis by adding to the existing pH depression caused by sulphate. Studies of this phenomenon by Galloway _et_ _al_. (1980) found that sulphate concentrations still exceeded nitrate on an equivalent basis, even during spring runoff (MOI, 1983).

The evidence and conclusions of published sources (Harvey _et_ _al_., 1981; Overrein _et_ _al_., 1980) lead to the conclusion (MOI, 1983) that for surface water systems, most of the increases in acidity observed was due to the changes noted in SO_4^{2-} concentration attributed to sulphate and sulphuric acid deposition. Both sulphuric and nitric acids contribute acidity to surface waters during periods associated with pH depressions and fish stress. However, the authors felt there was no strong evidence at present for anticipating any appreciable reduction in long-term lake or stream acidification from a reduction in nitrate inputs.

Automotive emissions represent one source of nitrate deposition, but are not related to the dry deposition of ammonium ion (NH_4^+) which is the other main atmospheric nitrogen component. As noted in Section 4.4.2, assimilation of nitrate by vegetation produces a net surplus of alkalinity, while the oxidation of NH_4^+ to NO_3^- and the taking up of NH_4^+ by plants are net producers of acidity.

4.5 Man-Made Materials

4.5.1 Introduction

Material damage related to air pollution has been recognized for well over 300 years (Yocom et al.,1982). Table 4.5-1 based on a review by Yocom and Upham (1977) summarizes the types of damage that may be caused by air pollution. The important points to be brought out from Table 4.5-1 are:

1. A variety of materials are damaged by air pollutants.
2. Air pollution is only one of many environmental factors that can cause damage to materials exposed to outdoor atmospheres.
3. Sulphur oxides figure prominently as an air pollution type which may cause damage to materials.

There are several fundamental problems in quantifying the extent of damage to materials form specific pollutants (Yocom et al.,1982).

1. Types of damages associated with air pollutants tend to occur in clean atmospheres and cannot be distinguished from those caused or enhanced by air pollution.
2. Laboratory studies are not representative of real life exposure.
3. Damage measured today may have been the result of higher air pollution levels in the past.
4. Changes in types of materials and protective coatings due to technological change have occurred.
5. Determination of the quantities of materials in place in relation to air pollutant exposures is difficult.

This chapter relies heavily upon materials effects section of U.S. criteria documents (EPA 1982a; EPA 1982b; EPA 1978) and review articles (Yocom et al., 1982; Lodge et al., 1981). In view of the foregoing, there is no ambient air standard in place today that has used the effects on materials as its basis.

TABLE 4.5-1

AIR POLLUTION DAMAGE TO MATERIALS

Materials	Types of Damage	Principal Air Pollutants	Other Environmental Factors
Metals	Corrosion, tarnishing	sulphur oxides and other acid gases *	moisture, air, salt microorganisms
Building stone	Surface erosion and discolouration	sulphur oxides, and other acid gases, particulate matter	moisture, temperature salt, vibration, microogranisms, CO_2
Paint	Surface erosion and discolouration	sulphur oxides, ozone, particulate matter	moisture, sunlight microorganisms
Textiles	Reduced tensile strength, soiling	sulphur oxides, nitrogen oxides particulate matter	moisture, sunlight physical wear
Textile Dyes	Fading, colour change	nitrogen oxides, ozone	sunlight
Rubber	Cracking	ozone	sunlight, physical wear
Ceramics	Changed surface appearance	acid gases, Hr & HF	moisture, microorganisms

underlined air pollutants of relevance to this study

Adapted from Yocom and Upham, 1977

* a gas when combined, or reacted with water forms an acid

4.5.2 Nitrogen Dioxide

Nitrogen oxides (principally NO_2) are capable of damaging several types of materials. The most significant effect is on certain types of fabric dyes. NO_2 is also involved in the weakening of some fabrics, causing deterioration of certain types of plastic materials, and in the corrosion of metals. Nitrogen oxides also play an indirect role in material damage by other pollutants such as O_3 and SO_2. Participation of NO_x in photochemical reactions results in the formation of ozone and in the photooxidation of SO_2 in the presence of reactive hydrocarbons to produce sulphuric acid aerosols (NAS, 1976).

The role of nitrogen dioxide in damaging fabrics and fabric dyes must be considered in conjunction with their exposure. Most garments spend most of their useful lives in indoor environments where NO_2 levels are different than in ambient air. In homes with no gas appliances NO_2 levels indoors are less than outdoors. Yocom et al. (1977) showed that concentrations of NO_2 inside homes with gas cooking can be several times outdoor concentrations, but that the concentration gradient from the kitchen to other parts of the home is extremely steep because of the short half life of NO_2 in homes. In fabric and clothing warehouses, indoor levels of NO_2 could be elevated if unvented heaters and combustion powered fork-lift trucks are used. Yocom et al. (1982) also caution against the use of chamber data to establish dose/reponse relationship because the complicating effects of other pollutants and wide variations in other environmental factors such as temperature and humidity are neglected. Thus chamber studies designed to determine the effects of a pollutant on material in which the presumed outdoor concentrations are being depicted tend to be misleading.

Dye Fading: Textile dye fading attributed to NO_x action includes fading of dyes on cellulose acetate, cotton, viscose rayon, and nylon; colour changes on permanent press garments; and yellowing of white fabrics.

The NO_2 fading of acetate dyed blue, or in shades in which blue is a component, results in pronounced reddening. The susceptible blue dyes are derivatives of anthraquinone. Blue dyes, such as Disperse Blue 3, are commonly used to test for the presence of NO_2. The fading on this dye is caused by the formation, through oxidation, of nitrosamine or phenolic groups, both of which have a red colour. Salvin et al (1952) found that cellulose acetate is an excellent absorber of NO_2. Absorption characteristics are believed to play an important role in dye fading mechanisms. Dye fading associated with NO_2 exposure of cellulose acetate and cellulosics summarized by US EPA (1982) is given in Table 4.5-2. Chemical changes, such as those cited in consumer complaints of dye on cellulose acetate, cotton and rayon, can take place within 3 months at NO_2 concentrations of 380 $\mu g/m^3$ (0.2 ppm) (Hemphill, 1976). Also, as shown in Table 4.5-2, the addition of SO_2 appears to accelerate the fading by NO_2 even though SO_2 by itself produces no change.

The investigations by Beloin in the field (1972) and laboratory (1973) show that at NO_2 concentrations of about 94 $\mu g/m^3$ (0.05 ppm) representative dyes for cotton and rayon will suffer serious fading (Table 4.5-3). Other fading effects noted on cotton/rayon fabrics are also summarized in Table 4.5-2. Upham et al (1976) carried out a chamber study of the effect of atmospheric pollutants on selected drapery fabrics. Fabrics were exposed to 0.05 and 0.5 ppm each of SO_2 (130 and 1300 $\mu g/m^3$), O_3 (100 and 1000 $\mu g/m^3$) and NO_2 (94 and 940 $\mu g/m^3$) under xenon arc irradiation at various humidities. The effect of NO_2 was pronounced, especially to vat-dyed drapery fabric. The most noticeable colour changes were at 940 $\mu g/m^3$ (0.5 ppm) and 90% relative humidity for a vat-dyed drapery fabric.

TABLE 4.5-2

FADING OF DYES ON CELLULOSE ACETATE AND CELLULOSICS *
(COTTON AND RAYON)

Dyed Fiber	Exposure	Pollutant	Concentration of Pollutant $\mu g/m^3$	Concentration of Pollutant ppm	Time	Effect	Reference
Acetate	Gas heated rooms	NO_2	3,760	2.0	N/A	Fading	Rowe and Chamberlain, 1937
Acetate	Chamber	NO_2	3,760	2.0	16 hr	Fading	Seibert, 1940
Acetate	Pittsburgh-Urban, Ames-Rural	NO_2-O_3	N/A		6 mo	Fading	Salvin and Walker, 1955
Acetate	Chamber	NO_2	3,760	2.0	16 hr	Fading	Salvin, et al., 1952
Cotton-Rayon	Clothes dryer	NO_2	1,128-3,760	0.6-2.0	1 hr cycle	Fading	Mclendon and Richardson, 1965
Acetate-Cotton, Rayon	Los Angeles[a]	NO_2	489	0.26	30 to 120 days	Fading	Salvin, 1964
		+ O_3	412	0.21			
		+ SO_2	131	0.05			
	Chicago[a]	, NO2	414	0.22			
		+ O_3	10	0.005			
		+ SO_2	655	0.25			
Cotton-Rayon	Chamber	SO_2-NO_2	3,760	2.0	16 hr	Fading	Salvin, 1969
Cotton-Rayon	Chamber	SO_2-NO_2 and 03	N/A		54 hr	Fading	Ajax et al., 1967
Range of fibers	Field-Urban, Rural	SO_2-NO_2+O_3	N/A		24 mo	Fading	Beloin, 1972
Range of fibers	Chamber	NO_2	94 to 940	0.05 to 0.5	12 wk	Fading	Beloin, 1973
Range of fibers	Chamber	NO_2 + Xenon arc radiation	940	0.5	10 to 80 hr	Fading	Hemphill et al., 1976
Acetate-Cotton, Rayon	Chamber	NO_2	94 to 940	0.05 to 0.5	N/A	Fading	Upham et al., 1976
Acetate-Cotton, Rayon	Survey	NO_2, SO_2, H_2S	Service Complaints		N/A	Fading	Upham and Salvin, 1975

* Reproduced from EPA (1982a)

TABLE 4.5-3

AVERAGE FADING OF 20 DYE-FABRIC COMBINATIONS[a] AFTER 12 WEEKS EXPOSURE TO NITROGEN DIOXIDE

Hunter Color Units*

			$94\ \mu g/m^3\ NO_2$				$940\ \mu g/m^3\ NO_2$			
Material	Dye	Color Index No.	Low Temp. Average 12.78°C	High Temp. Average 32.22°C	Low Humidity Average (50% RH)	High Humidity Average (90% RH)	Low Temp. Average 12.78°C	High Temp. Average 32.22°C	Low Humidity Average (50% RH)	High Humidity Average (90% RH)
Cotton	Direct	Red 1	7.2	8.0	7.4	7.8	18.0	20.4	16.1	22.3
Rayon	Direct	Red 1	3.4	T	T	T	13.4	16.3	12.6	17.0
Wool	Acid	Red 151	T	T	T	T	T	T	T	T
Cotton	Reactive	Red 2	T	T	T	T	10.4	6.9	9.7	7.6
Acrylic	Basic	Red 14	T	T	T	T	T	T	T	T
Cotton	Azoic[c]	Red	T	T	T	T	T	T	T	T
Nylon	Acid	Orange 45	5.6	17.0	10.1	9.5	21.5	27.9	24.3	25.1
Wool	Acid	Yellow 65	T	T	T	T	T	T	T	T
Acrylic	Basic	Yellow 11	T	T	T	T	T	T	T	T
Cotton	Sulfur	Green 2	T	3.3	T	T	6.5	6.6	6.1	7.1
Wool	Acid	Violet 1	T	T	T	T	T	T	T	4.3
Cotton	Direct	Blue 86	5.9	9.5	9.4	6.0	14.1	17.2	14.2	17.1
Cellulose Acetate	Disperse	Blue 3	29.0	42.3	37.7	33.6	86.9	75.6	80.0	74.4
Nylon	Disperse	Blue 3	5.5	14.7	5.9	14.2	39.6	45.5	34.0	51.3
Cellulose Acetate	Disperse	Blue 27	6.4	4.9	3.8	7.5	20.5	26.0	17.0	29.6
Polyester	Disperse	Blue 27	T	T	T	T	T	T	T	T
Cotton	Reactive	Blue 1	3.9	13.6	9.6	7.9	31.8	41.7	35.4	38.1
Cotton	Reactive	Blue 2	6.4	10.6	8.2	8.9	30.5	41.6	33.0	30.4
Cotton	Vat	Blue 14	6.3	6.7	3.3	9.7	34.3	30.4	23.4	41.3
Acetate	---[d]	AATCC Ozone Ribbon	T	T	T	T	5.7	11.7	5.7	11.7

[a] Each average, e.g. the low temperature average, was calculated by averaging the color change of duplicate samples from both the low temperature-low humidity and low temperature-high humidity exposure periods.

[b] T-trace (less than 3 units of fading). The higher the number, the greater the fading. Hunter Color Units approximate the NBS color scale.

[c] Coupling Component 2, Azoic Diazo Component 32

[d] C.I. Disperse Blue 27, C.I. Disperse Red 35, C.I. Disperse Yellow 37.

* Reproduced from EPA (1982a)

Test procedures for NO_2 which demonstrated the vulnerability of disperse dyes used on acetate, showed little change when the same dyes were used on nylon (Table 4.5-4). The fading of disperse blue dyes were shown to be due to ozone in the presence of high humidity (Salvin, 1974; cf Section 4.5.3). Acid dyes which are more resistant to O_3 were substituted as a remedial measure (US EPA, 1982). The vulnerability of acid dyes on nylon to NO_2 was noted (Imperial Chemical Industries, 1973) and dye manufacturers pointed out the importance of dye selection in carpets and home furnishing where high NO_2 levels might be expected.

The introduction of permanent press and double-knit garments, made of polyester were accompanied by cases of fading attributed to either NO_2 or O_3 (Table 4.5-4). The use of zinc nitrate catalyst instead of magnesium chloride has minimized migration of dyes to permanent press garment surfaces and made garments less susceptible to dye fading (Urbanik, 1974).

Yellowing of White Fabrics: In a number of circumstances, air pollutants have caused yellowing of white fabrics; NO_2 was established as the responsible pollutant. Table 4.5-5 presents a summary of the yellowing effects and concentration of NO_2. In all cases, yellowing occurred at a NO_2 concentration of 0.38 mg/m^3 (0.2 ppm), usually with high relative humidity.

Fabric Damage: Cotton and nylon fibre strengths are reduced by the hydrolytic action of acid aerosols. This problem assumes economic importance because industrial fabrics comprise a large end use of fibers, many of which are used in the production of cordage, belts, tarpaulins, and awnings (U.S. EPA, 1982). The products are exposed to air pollutants over long time periods outdoors. However, it has not been possible to isolate the effects of nitrogen oxides from that of sulphur oxides in field studies which have shown cotton fibre strength loss (Brysson _et al_., 1967; Brysson _et al_., 1968; Morris, 1966; Travoricek, 1966). The results for nylon are also inconclusive (Zeronian _et al_., 1971).

TABLE 4.5-4

EFFECT OF NITROGEN DIOXIDE ON FADING OF DYES ON NYLON AND POLYESTER *

Dyed Fibers	Exposure	Pollutant	Concentration of Pollutant µg/m³	ppm	Time	Effect	Reference
Nylon	Chicago Los Angeles	NO_2	188 282	0.1 0.15	30 to 120 days 30 to 120 days	Fading	Salvin, 1964
Polyester	Chicago Los Angeles	NO_2	376 282	0.2 0.15	30 to 120 days 30 to 120 days	Unchanged	Ibid.
Nylon	Urban Sites	NO_2	376	0.2	3 to 24 months	Fading	Beloin, 1972
Polyester	Urban Sites	NO_2	376	0.2	3 to 24 months	Unchanged	Ibid.
Nylon	Chamber High Humidity	NO_2	188 to 1,880	0.1 to 1	12 weeks	Fading	Beloin, 1973
Polyester	Chamber High Humidity	NO_2	188 to 1,880	0.1 to 1	12 weeks	Unchanged	Ibid.
Nylon	Chamber High Humidity	NO_2	376	0.2	48 hours	Fading	Imperial Chemical Industries, 1973
Nylon	Chamber High Humidity Xenon Arc	NO_2	940	0.5	30 to 120 hours	More fading than without NO_2	Hemphill et al., 1976
Polyester Permanent Press	Chamber	NO_2	940	0.5	16 hours	Fading	Salvin, 1966
Polyester Textured Double Knit	Chamber	NO_2	940	0.5	16 hours	Fading	Urbanik, 1974

* Reproduced from US EPA (1982)

TABLE 4.5-5

YELLOWING OF WHITES BY NITROGEN DIOXIDE*

Fiber	Exposure	Pollutant	Concentration of Pollutant		Time	Effect	Reference
			$\mu g/m^3$	ppm			
Survey	Service Complaints	N/A			N/A	Yellowing	Upham and Salvin, 1975
Rubberized Cotton	Chamber	NO_2	376	0.2	16 hr	Yellowing	Burr and Lannefeld, 1974
Rubberized Cotton	Chamber	NO_2	376	0.2	16 hr	Yellowing of anti-oxidant	Salvin, 1974c
Spandex	Chamber	NO_2	376	0.2	8 hr	Action on fiber	Ibid.
Acetate Optical brightener	Chamber	NO_2	376	0.2	8 hr	Yellowing	Ibid.
Nylon Optical brightener	Chamber High Humidity	NO_2	376	0.2	16 hr	Yellowing	Ibid.
Nylon Anti-stat finish	Chamber High Humidity	NO_2	376	0.2	16 hr	Yellowing	Ibid.
Cotton Cationic softener	Chamber	NO_2	376	0.2	16 hr	Yellowing	Ibid.

* Reproduced from US EPA (1982)

Corrosion of Metals: Nitrogen oxides, as potential precursors to acid pollutants are implicated in several types of corrosion: pitting, selective leaching, and stress corrosion. However, it is believed that their role in outdoor corrosion of metals is much less important than sulphur oxides (Yocom *et al*.,1982). The US EPA (1982) has summarized the principal research results of nitrogen oxides and nitrates in qualitative terms (Table 4.5-6). The rusting process is the formation of an iron oxide which acts as a protective film. When an acid aerosol is present, this protective layer can be broken down and new surfaces exposed to corrosion. A liquid film or the presence of a hydrated salt plays an important part in corrosion.

Unfortunately in assessing the effects of air pollution on metals, work has stressed SO_2 as the major causative agent (Gillette, 1975) although NO_2 is abundant in urban atmospheres. Sulphur oxides and relative humidity have been documented as the most important factors for producing corrosion (Haynie et al, 1976; Yocom and Grappore *et al*.. 1976). Investigations in California did find that the cause of premature failure of nickel/brass springs in telephone equipment was due to a dust rich in nitrates (Hermance *et al*., 1971). Nitrate deposition correlated with relay failure, but no relation to the concentration of NO_2 in air was established. The important finding in this study was that nitrate salts were found to be more hygroscopic than chloride and sulphate salts. Consequently, nitrates may lower the threshold humidity requirements for formation of a liquid film which can promote corrosion. Hermance (1966) also reported on the failure of telephone switches by "creeping green corrosion" in various United States cities. It was found that nitrate deposits greater than 15.5 $\mu g/cm^2$ were formed on the nickel bases of palladium-capped contacts and that these deposits gradually crept over the contact. This corrosion effect may be a result of lowering of the threshold humidity requirements (NAS, 1976) by nitrates.

TABLE 4.5-6

CORROSION OF METALS BY NITROGEN DIOXIDE *

Metal	Exposure	Pollutant	Effect	Reference
Mechanics of Corrosion - Function of Nitrates				National Academy of Sciences, 1976a
Nickel Brass	Los Angeles	Nitrates	Strength Loss	Hermance et al., 1971
Nickel Brass	Los Angeles	Nitrates	Strength loss	McKinney and Hermance, 1967
Nickel	Los Angeles New York	Nitrates	Corrosion	Hermance, 1966
Tungsten	Chamber	NO_2	Change oxide surface	Lazareva, 1973
Electronic contacts	Field	NO_2-SO_2-H_2S	Corrosion film	Chiaranzelli and Juba, 1966
Metal parts	Field	NO_2-SO_2-O_3	Failure	Gerhard and Haynie, 1974
Economic Costs of Corrosion				Fink et al., 1971

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* Reproduced from US EPA (1982)

Gerhard and Haynie (1974) examined the causes of catastrophic failure of metals leading to loss of life as well as collapse of the metal structure. Their conclusion was that air pollutants were a probable partial causative factor but they did not determine the relationship between levels of particular air pollutants and metal failure. All of the above studies are summarized in Table 4.5-6.

The National Academy of Sciences (1976) concluded upon their review of the literature that evidence exists that nitrogen can affect the rates of oxidation of metals and alloys and that nitrate absorption of water moisture assists in the formation of a liquid film which promotes corrosion. However, there is no direct relationships between NO_x or NO_3 levels in ambient air and corrosion behaviour.

Other Effects: Chamber exposures of polyethylene, polypropylene, polystyrene, polyvinyl chloride, polyacrylonitrile, polyamides and polyurethane plastics to combinations of SO_2, NO_2, and O_3 have resulted in deterioration. NO_2 alone has caused chain scission, which results in lower strength in nylon and polyurethane at concentrations of 1.88 to 9.40 mg/m^3 (1.0 to 5.0 ppm).

In summary, the damaging effects of NO_2 on fabric dyes have been well established for a number of textile and dye combinations. Yellowing of white fabrics by NO_2 has also been established. In applying the damage level concentrations for dye-fading and yellowing, one must take into account indoor exposure as discussed at the beginning of this section. Information as to the contribution of NO_2 to textile degradation is incomplete as is the data on metal corrosion. In the latter case, the extensive data available relate corrosion data to SO_2 levels, although NO_2 may have an effect by lowering the humidity threshold for corrosion.

4.5.3 Photochemical Oxidants (Ozone)

This section addresses ozone effects on fabrics, dyes, elastomers (rubber products), as well as some possible other effects. The material was adapted mainly from the U.S EPA critera document (1978), the National Academy of Science review document (1977), the review of air pollutant effects on materials by Yocom et al (1982), and is supplemented by recent publications (citations supplied by B. Tilton, US EPA, 1978).

Fabrics: In general the degradation of fabrics has not been well quantified. Bogarty et al (1951) concluded that effects of other factors such as sunlight, heat, wetting and drying, and micro organisms far outweigh the effects of ozone on cotton duck and print cloth. Kerr et al. (1969) do indicate that a synergistic effect of moisture and ozone may be a contributor to cotton material degradation but these experiments were at ozone levels of 2 mg/m^3 (1000 ppb) far above ambient levels in Canada. Zeronian (1971) indicates little if any effect of ozone on synthetic fibres. Based on these studies the National Academy of Sciences (1977) expressed the view that ozone actually has little if any effect on textiles, fibres, and synthetic cloth exposed outdoors.

Dye Fading: In Section 4.5.2 it was indicated that NO_2 may cause dye fading. Ozone can produce similar effects. The same points about indoor exposure made in that section (from Yocom et al., 1982) about indoor exposure also apply to O_3. In addition for ozone, there are few indoor sources, and since ozone reacts rapidly with indoor surfaces, inside concentrations are usually a very small fraction of those outdoors. The effects of ozone on dye-fading have been termed O-fading. For dye-fading, the colour change is a complex function of ozone and other gaseous pollutants, moisture, dye composition, fibre composition, compactness of the fibre structure, and the accessibility of dye to ozone. In the past, permanent press garments made of polyester and cotton as well as nylon capacity were the main products affected (Salvin, 1969). Remedial measures to correct the permanent press problem have included selection of dyes more resistant to reaction with 0_3 and NO_2 and replacement of the magnesium chloride catalyst used in the permanent press process with a zinc nitrate catalyst. Ozone fading most affected disperse dyes of the anthraquinone series, but not the azo series of dyes. Ozone concentrations of 200 $\mu g/m^3$ (100 ppb) produced marked fading in most of the blue disperse dyes and in some reds and yellows. The choice of catalyst plays an important role as the migration of disperse dyes increases significantly when magnesium chloride is used rather than zinc nitrate.

Haylock and Rush (1976) studied ozone fading of anthraquinone dyes on nylon fibres (carpet yarn) in controlled environments. The fading curves were highly consistent with Upham et al's (1976) assumed relationship for NO_2:

$$\Delta E = \Delta E_m (1 - \ell^{-at})$$

where ΔE = amount of fading, fading units
ΔE_m = maximum fade
t = time in years
a = constant containing the effects of environmental factors

Regression equations on the Haylock and Rush (1976) data account for 99 percent of the variability. For exposures at 40°C and 90% RH, the results are as follows:

Olive 1 anthraquinone dye: $a = 0.38+3.65\text{x}10^{-5}O_3$
Olive II anthraquinone dye: $a = 0.00107+1.18\text{x}10^{-6}O_3$
C.I. Disperse Blue 7 dye : $a = 0.212+5.35\text{x}10^{-6}O_3$

where O_3 is in $\mu g/m^3$ (e.g. 2 x ozone concentration in ppb). Percentage life lost can be expressed by the following:

Olive 1 : $$\text{Percent Life Lost} = \frac{0.096\ O_3}{1 + 0.000960\ O_3}$$

Olive 11: $$\text{Percent Life Lost} = \frac{1.1\ O_3}{1 + 0.011\ O_3}$$

C.I. Disperse Blue 7: $$\text{Percent Life Lost} = \frac{0.025\ O_3}{1 + 0.00025\ O_3}$$

These relationships curve only slightly below ozone levels of 100 $\mu g/m^3$ (50 ppb). The costs of early replacements can be assumed to be directly proportional to ozone lives. Kamath et al. (1982) also studied the effect of O_3 on dye fading (C.I. Disperse Blue Dye 3) on nylon fibres. The fibres were exposed to ozone concentrations, of 400 $\mu g/m^3$ (200 ppb) at 40°C and relative humidities of 90 percent, 85 percent and 65 percent. The results of their laboratory studies indicated that relative humidity has a significant effect on fading, that destruction of the dye begins near the fibre surface, and that with long exposure penetration into the fibre may be an important mechanisms in ozone fading. At 65 percent relative humidity, the fading rate dropped to very low levels. Thus Haylock and Rush's (1976) relationships are only applicable to 90% ralative humidity.

Nipe (1981) summarized the results of a 3-year study designed to establish the relationship between in-service atmospheric contaminant fading by ozone of carpets in a home. Test carpets consisted of nylon 6 and 66 dyed with two disperse and two acid dye formulas. Statistical analyses of the data showed no correlation between outside weather conditions and in-house fading by ozone or NO_2. For all samples, much greater fading did occur during the summer than in winter. Typically ozone levels are higher during summer months, and doors and windows are open more during this seasons thus allowing a greater exchange between inside and outside air.

No general conclusions can be made about ozone fading other than that there is an effect for certain fibres and dyes under selected conditions (e.g. Haylock and Rush's relationships). Each dye has its own sensitivity to ozone effects and other environmental factors and no correlation appears to exist between indoor exposure and outdoor ozone concentrations. The replacement of the permanent press magnesium chloride catalyst with zinc nitrate probably prevents dye deterioration sufficiently to not decrease the useful life of clothing items.

Elastomers: Natural rubber and some synthetic elastomers account for the bulk of outdoor exposed elastomeric products (Mueller and Stickney, 1970). It has long been known that certain types of rubber compounds, especially natural rubber, are particularly susceptible to ozone attack (Cotton, 1937; Bradley and Hagen-Smith, 1957). At pollutant concentrations and stress levels normally encountered in outdoor environments, the elastomer hardens or becomes brittle and cracks propogate through it resulting in a loss of physical integrity. Several factors influence the action of ozone on elastomers:

(i) nature of the elastomeric material
(ii) degree of stress
(iii) ozone concentration and exposure
(iv) temperature and pressure
(v) presence of inert fillers and antiozonants

Elastomers sensitive to ozone attack contain olefin structures (Barley, 1958). Natural rubber, styrene-butadiene, polybutadiene, and polyisoprene contain these chemical structures. Styrene-butadiene lattices are used for tire-cord dipping and styrene-butadiene rubber is used to manufacture products such as wire and cable coatings, footwear, tires, and belts and hoses (CPI Product Profiles, 1981). Polybutadiene is used in the manufacture of tires (ibid, 1981). Polyisoprene is the synthetic equivalent of natural rubber and is used in the manufacturing of tires, footwear, wire and cable covering, and footwear (ibid, 1981). One of the major applications of natural rubber and polyisoprene has been in the building and recovery of truck and off-road tires. A typical bias-ply passenger tire uses

78% synthetic and only 22% natural rubber. In contrast, radials employ only 52% synthetic but 48% natural rubber.

Two effects can occur as a result of the ozone-olefin reaction. One is chain scissioning which decreases the molecular weight and the tensile strength. The other is crosslinking which causes the elastomer to become brittle.

Items mentioned such as wire and cable, hose, footwear, tires are under stress and in fact any elastomeric object that is flexed or folded produces a stress. If no stress is applied, elastomers can be exposed to high ozone concentration without the formation of cracks. However, when natural rubber stressed as little as 2-3% in extension is exposed to 22 $\mu g/m^3$ (11 ppb) of ozone, surface cracks are observed at right angles to the stress direction (Crabtree and Malm, 1956).

The initiation of cracking is controlled by the dose of ozone (concentration x time). Bradley and Hargen-Smit (1951) found that when strained natural rubber strips were exposed to 40,000 mg/m^3 (20,000 ppm) ozone, specimens cracked within 1 second. When the rubber was exposed to lower concentrations of ozone, longer time periods were required to develop cracks.

Various research indicates that the rate of ozone attack is approximately doubled with every 10°C increase in temperature (Braden and Gart, 1960; Crabtree and Malm, 1956; Jaffe, 1967). Many laboratory experiments are conducted at a temperature of 30°C or higher. Veith and Evans (1980) found that a change in pressure due to elevation will increase the rate of cracking by as much as 16% at higher pressure.

There are two main approaches to avoiding ozone damage - addition of antiozonants and use of waxes or other protective coatings. The antiozonant action is not well understood chemically, but probably involves formation of a protective film which slows crack growth rates. The more extensively used antiozonants are located in Table 4.5-7.

TABLE 4.5-7
ELASTOMER ANTIOZONANTS

N,N' - Diphenyl-p-phenylenediamine
N,N' - Di-(2-Octyl-)-p-phenylenedimine
N,N' - Di-2-(5-Methylheptyl)-p-phenylenediamine
N,N'bis(1,4-Dimethylphentyl)-p-phenylenediamine
N,N'bis(1-Ethyl-3-methylpentyl)-p-phenylenediamine
N,N'bis(1-Methylheptyl)-p-phenylenediamine

Dose-reponse relationships for expcsed elastomeric materials have been developed, unfortunately most of the work has involved high ozone levels and rubber formulations without antiozonants. Hence most of the results do not apply to urban environments. Some relatively recent studies examine tire products protected by antiozonants.

Haynie et al. (1976) conducted tests on white sidewall specimens from a steel-belted radial tire. The level of ozone was found to be a statistically significant factor in the rate of cracking of the white sidewall rubber. Haynie concluded that even at 160 $\mu g/m^3$ (80 ppb) average ozone concentration it would take 2½ years for a crack to penetrate to cord depth. Therefore, it is unlikely that sidewall failure from ozone damage would cause reduced time life.

Gandslandt and Svensson (1980) tested the decrease of isoelastic force on exposure to ozone ten different mixtures rubber compounds, composed of natural rubber (NR), and 2 synthetic rubbers. The samples were exposed to concentrations of 10 mg/m^3 (5000 ppb) at 30°C. The compounds with antiozonant protection showed greatest resistance to effects of ozone while those compounds protected only by paraffin wax, demonstrated the least resistance to ozone attack.

Davies (1979) evaluated the effects of ozone and sunlight on interply adhesion on uncured rubber. Excellent adhesion of plies is essential in the manufacture of tires, otherwise product strength is reduced. Prior to cure, Davies' test results indicate that adhesion of Styrene-butadiene rubber (SBR) compounds is unaffected by exposure to ozone concentrations of 30 mg/m^3 (15 ppm), while an NR/SBR blend showed a 30 percent decrease in adhesion. Large reductions in adhesion, on the order of 70%, occurred with NR compounds. Davies also examined the role of antioxidants, antiozonants and waxes. Only the fast blooming waxes were found to afford protection against ozone. After exposure to sunlight alone, the antioxidants, in general, functioned properly in maintaining good adhesion, while the waxes gave only fair protection. Davies concluded that to protect NR from O_3 attacks as well as sunlight, both wax and antioxidants or antiozonants are required. Similar effects of poor adhesion of resorcinal-formaldehyele latex dipped tire cords were noted by Wenghoefer (1974).

For air pollutant induced damage to be economically important, the useful life of the product must be significantly affected by ozone exposure. The useful life of most rubber products is probably determined by normal use rather than pollutant damage (EPA, 1978). Vehicle tires have been identified as the only major use of rubber where economic costs result from the effects of ozone (Stankunas et al., 1982). The additional costs are mainly attributed to antiozonant protection.

Paints: The primary effect of ozone on paint is accelerated erosion of the paint film. Weight loss converted to thickness loss over a specified exposure period is the usual method of measuring this effect on test panels. This enables a prediction of the life of the panel through extrapolation of exposure data.

In the laboratory work of Campbell et al. (1974) exposures, both shaded and unshaded at 2000 μg/m^3 (1000 ppb), produced measurable effects with oil based paint showing the greatest effect. Exposure at 200 μg/m^3 (100 ppb) did not produce statistically significant erosion rates. Spence et al. (1975) developed a damage function for acrylic coil coating which is somewhat academic because at an average level of 100 μg/m^3 (50 ppb) O_3 the relation predicts a 20 μm film would last over 80 years. Mansfield's (1980) work indicates that there may be a statistically significant relationship between the erosion of latex paint and relative humidity and ozone; however, further studies are ncessary before a cause and effect relationship can be established. Also there are no available data to address potential synergistic effects of ozone in conjunction with other pollutants such as nitrogen dioxide and/or sulphur dioxide.

Effects on Other Materials: The NRCC (1975) review of the effects of photochemical smog on other materials included possible effects on plastic and asphalt. However, these effects have little direct applicability since they were recorded in a laboratory environment at ozone levels extremely higher than ambient concentrations. Haynie and Upham (1971) reported a possible beneficial effect of photochemical oxidants on inhibition of steel corrosion in the field but laboratory studies and theory did not confirm the effect. Polyethylene, an electric insulating material, may be adversely effected by ambient ozone (NAS, 1977) but quantitative data is lacking; there is no evidence that the chemical reactions go far beyond the surface. The NAS document cites that expert opinion believed that ozone effects on polyethylene insulation and other polyethylene products are negligible compared with the embrittlement of polyethylene by a combination of oxygen and solar ultraviolet radiation.

In summary the only demonstrable and potentially quantifiable effects of ozone at ambient levels are associated with tire products. Here the measure of the effect is the amount of antiozonant substance that must be added by a manufacturer to prevent degradation of the product. There are effects on dye fading and possibly erosion of latex house paint but these effects are presently not quantifiable, except in controlled circumstances under a very limited range of environmental factors.

4.5.4 Diesel Exhaust Particulates

Diesel exhaust particulates are composed mainly of soot (Amann and Siegla, 1982) in the fine particle size range. Damage to building materials by airborne particles is basically by soiling; however no statements of acceptable particulate levels with regard to soiling have been documented (Lodge et al, 1982). Damage to paints or appearance degradation by particulate pollutants is suspected, but other factors such as improper surface preparation and exposure to sunlight are probably more important. A loss of function of high-voltage insulators due to particles (both natural and man-made) is a problem (Gijalva and Talamas, 1979) but is mitigated by over-insulation, greasing, cleaning, and other measures. The physical processes involved and the composition of deposits are far from being understood.

Reidere (1974) indicated that much serious destruction of stone monuments in Germany is due to natural weathering rather than to air pollutants. He did also cite examples of serious damage to sculptured stone by soot. Thick crusts of soot form in areas protected from the washing of rain. Although this effect has been documented no quantitative dose-response exist.

In summary, there is no scientific data base available for quantitatively determining the effects of particles on materials in general and consequently no data applicable to fine particle soot fractions.

4.5.5 Summary

Damage to man-made materials by ozone and nitrogen dioxide have been documented at levels experienced in the ambient atmosphere. Fading of garments must consider indoor exposure, or levels given in this chapter may be misconstrued.

Cellulosic fibres (cotton and viscose rayon) dyed with direct dyes, vat dyes, and fibre reactive dyes, suffer severe fading on chamber exposures to 940 $\mu g/m^3$ (0.5 ppm) NO_2 with high humidity (90%) and high temperatures (30°C). Significant fading is observed in 12 weeks exposure to 94 $\mu g/m^3$ (0.05 ppm) under the same high humidity and temperature conditions. Acid dyes on nylon fade on exposure to NO_2 at levels of 188 $\mu g/m^3$ (0.1 ppm) under the same environmental conditions. Yellowing of white fabrics for polyurethane segmented fibres, rubberized cotton, optically brightened acetate, and nylon have been documented in chamber studies using 376 $\mu g/m^3$ (0.2 ppm) NO_2 for exposures of 8-hours. Nylon may suffer chain scission when exposed to 1.88 to 9.4 mg/m^3 (1.0 to 5.0 ppm) NO_2.

Nitrates have been implicated in the cracking of wires made of nickel brass alloy used in telephone equipment but no other evidence exists for metal corrosion. Since nitrate salts are more hygroscopic than chloride or sulphate salts, nitrates may lower the threshold requirements for wet metal corrosion.

The only quantified effects of ozone on materials are for tire products. Stressed natural rubber cracks at ozone concentration as low as 40 $\mu g/m^3$ (20 ppb). Tires are protected from ozone degradation by the addition to antiozonants which inhibit ozone reactions. There are effects of ozone on textile dye fading under select condition which cannot be generalized to the urban environment. All effects occurred at high temperatures and high relative humidities. Some recent work by Mansfield (1980) has indicated there may be a significant association between the erosion of latex paint and relative humidity and ozone but further work is required to provide a definitive dose-response damage function.

4.6 Visibility

4.6.1 Introduction

Visibility may broadly be defined as the degree of clarity of the atmosphere. The World Meteorological Organization (1971) defined meteorological visibility as "the greatest distance at which a black object of suitable dimensions, situated near the ground, can be seen and recognized, when observed against a background of fog or sky". Although visibility is commonly expressed in terms of visual range as above, it is really more than being able to see a black target at a certain distance. Visibility also includes seeing targets at shorter distance than the visual range and being able to appreciate the target details such as colour and texture. People comment on how hazy it looks or how clear it is, the brightness of colours or the brownish or bluish colour of air. For example, the City of Calgary on occasion has a shroud of brown or white haze which occasionally covers the city and obscures the view of the Rocky Mountains (Harrison and Mathai, 1980). Thus, visibility is in the most part an aesthetic value, although atmospheric parameters may be used to obtain a physical measure of visual air quality such as range or colour. One clear effect of reduced visibility is the transistion from visual flight rules (VFR) to instrument flight rules (IFR) by aircraft when the visual range decreases below 1.6 kilometres in uncontrolled airspace and below about 5 kilometres in controlled airspace. One of the criteria used in setting the Canadian maximum air quality objective for suspended particulates was visibility reduction below 8 km (Subcommittee on National Air Quality Objectives, 1976).

Visual air quality is probably the first indicator by which an average person becomes aware of air pollution. Preliminary studies of social awareness/perception and the economic value of visibility in cities and rural areas indicate that visibility is an important aesthetic value in both settings (EPA, 1982; MOI, 1983). A study by Flachsbart and Phillips (1980) in Los Angeles indicated only two air quality indices, ozone and visibility, were consistently significantly related to perceived air quality. While air pollution has its greatest visibility effect near its source, it can affect visibility hundreds of kilometres away. In this respect, visibility degradation from the natural state, like acid precipitation, may be considered as a potential problem arising from the long range transport of pollutants (MOI, 1983). Evidence of extensive hazy air masses ($>10^5$ km^2) over land and sea have been observed from satellites (Lyons and Pearse, 1976). The widespread dispersion of man-made emissions and visibility reduction has prompted the United States to consider visibility related standards.

There are five broad categories of visibility impairment which can be ascribed in all cases to the presence in air of particles, aerosols, or gases:

(i) visible plumes (plume blight) from large industrial sources
(ii) urban scale visibility impairment characterized by loss of contrast, decreased visual range and a "whitening" or "browning" effect
(iii) regional scale (several hundred kilometres) low to medium relative humidity hazes
(iv) wind blown dust in agricultural areas
(v) fog and high relative humidity haze which may be of natural origin or may be induced by the presence of man-made emissions

Precipitation, drifting snow, and sky cover which limit visibility have not been mentioned in the above categories because they are strictly of natural origin. In each of the given categories, visibility limitations could occur naturally, but air pollution can aggravate the problem. For the purposes of this report on the effects of automobile emissions, categories (ii), (iii) and (v) are especially relevant. Visibility impairment or degradation will be considered as any visually perceptible change in visibility from that existing in clean air. The emphasis is on both distance through the atmosphere at which objects can be seen and discolouration.

Variations in visibility are basically governed by the concentration and nature of particles in air. Gaseous NO_2 plays a minor role, but it may be of importance in colouration of a polluted atmosphere. Particles in air reduce visibility by scattering and absorbing light, while gases such as NO_2 absorb light. Light scattering by particles is dominated by the size range 0.1 to 2.0 μm diameter. Secondary particle products (formed through chemical reaction), such as SO_4 and NO_3 are especially important because they tend to accumulate in this size range and attract water vapour into the aerosol phase. Elemental carbon (black soot) is the most important type of light absorbing particle. The emissions of most relevance to visibility are sulphur dioxide (precursor of SO_4), NO_x (precursor of NO_3 particles and gaseous NO_2), HC (precursor of secondary organic particles), and soot.

Before discussing the effects on visibility of the various constituents, the various mathematical relationships describing visibility are presented.

Atmospheric visibility depends on light transmission through air and the eye's ability to identify an object by its contrast with the background. Contrast is mathematically defined as the ratio of object brightness minus horizon or background brightness to horizon (background) brightness:

$$C = \frac{B_o - B_h}{B_h} \qquad (1)$$

Scattered and absorbed light reduces the contrast of an object with its background (Figure 4.6-1). The combined effects of scattering and absorption are referred to as extinction and are represented by an extinction coefficient, b_{ext}. This coefficient is further broken down into absorption and scattering components:

$$b_{ext} = b_{ag} + b_{rg} + b_{ap} + b_{sp} \qquad (2)$$

where b_{ag} = absorption by gases (usually small in rural areas)

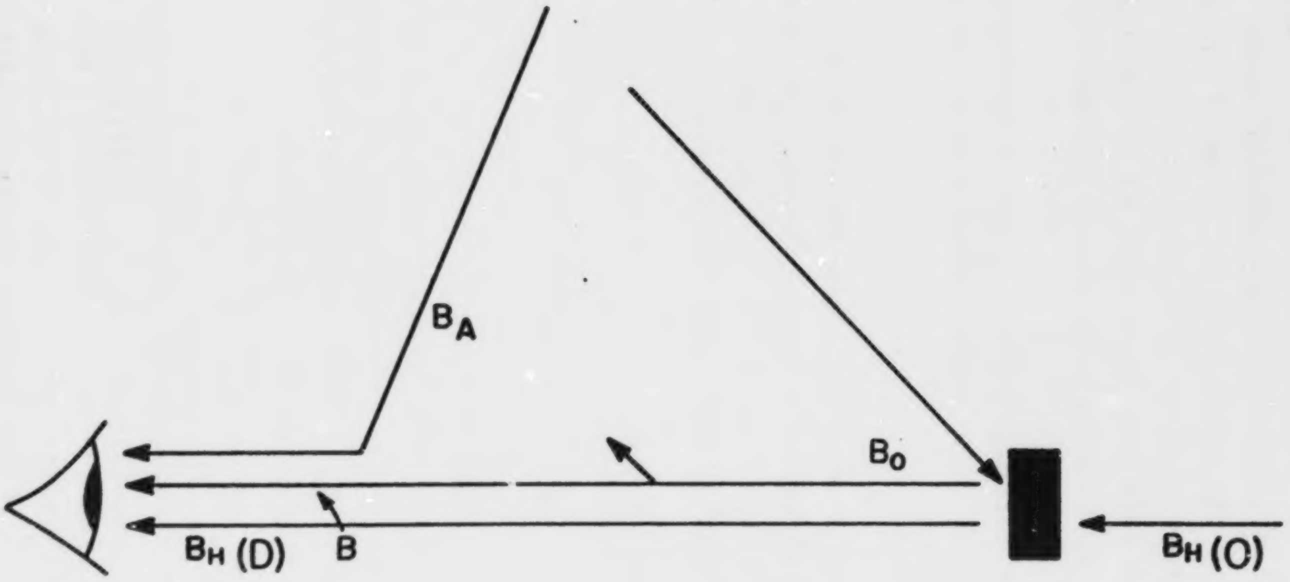
b_{ap} = particle absorption coefficient (units in inverse distance; e.g. km^{-1} may be large in urban areas)

b_{rg} = Rayleigh or blue sky scattering ($\simeq 0.12\ km^{-1}$)

b_{sp} = particle scattering coefficient (always fairly large in urban and rural settings)

Some indication of the relative importance of each of the coefficients,

Figure 4.6-1 Contributions to luminosity seen by an observer. B_H is background brightness, B_o is target brightness and B_A is "airlight" brightness.



Adapted from NRCC (1982)

which specify the rate at which a beam of light is attenuated as it travels through air, has been given in the description of the four parameters in equation (2).

Through the Koschmeider equation, with b_{ext} measured or estimated from fine particle concentrations, visual range may be estimated by:

$$V = \frac{K}{b_{ext}} \tag{3}$$

where V = visual range (distance at which black object is just visible against the sky)

b_{ext} = as defined in equation (2)

K = a function of the intrinisic target brightness and observer threshold contrast which is a function of the observer and target size

For a contrast threshold of 0.02 and a typical observer, K is usually assumed to be 3.9. The scattering portion of extinction has typically been correlated on the order of 0.9, with visual range (EPA, 1981). Empirical determinations involving the Koschmeider relation have yielded lower values than the assumed 3.9. The most complete analysis (Ferman et al., 1981) reported a value of 3.5 for well mixed periods; other researchers' values have ranged from 1.7 to 3.6.

4.6.2 Gases - Nitrogen Dioxide

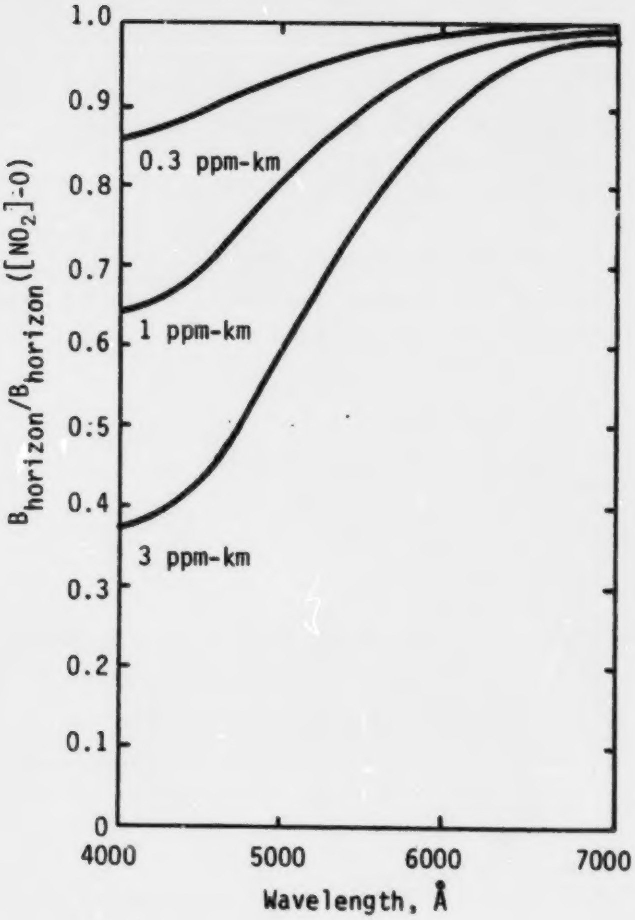
This section has largely been adapted from the final draft U.S. EPA NO_x criteria document (1982). Nitrogen dioxide absorbs light particularly at the shorter wavelengths - violet, blue, and green, reducing the brightness and contrast of distant objects and causing the sky horizon and white objects to appear reddish-brown (NAS, 1977). The discolouration of the horizon sky due to NO_2 absorption is determined by the relative concentrations of NO_2 and light-scattering particles. Independent of NO_2 absorption, wavelength-dependent scattering by small particles can also produce a noticeable brown colour (Husar and White, 1979). In addition, particle light scattering is known to be the primary cause of visibility reduction in photochemical smog situations with NO_2 light absorption playing a minor role (Charlson et. al., 1969; Wolff *et al.*, 1982).

The brightness of the horizon sky in the presence of NO_2 and light scattering particles can be described by the following relationship (Robinson, 1977):

$$\frac{B_h}{B_{ho}} + \frac{b_s}{b_{ext}} = \frac{b_{sp}}{b_{sp} + b_{ag}} = \frac{1}{1 + \frac{b_{ag}}{b_{sp}}} \qquad (4)$$

where B_h and B_{ho} are the brightness of the horizon sky with and without NO_2, and b_{ag} refers to absorption by NO_2. At 550 nm, b_{ag} x 10 is equal to 3.3 x NO_2 where NO_2 is in units of ppm and b_{ag} in km^{-1} (Hodkinson, 1966). As previously indicated, visual range is inversely proportional to b_{ext}, which in many cases is dominated by particle scattering (b_{sp}). Since b_{ag} is proportional to the concentration of NO_2 in air, the ratio b_{ag}/b_{sp} is proportional to the product of NO_2 concentration and visual range. The calculated alteration, contributed by NO_2, to horizon brightness in the presence of particle scattering is given in Figure 4.6-2. This calculation neglects the wavelength dependence of scattering, which can be substantial in relatively clean air and mitigates the discolouring effects of NO_2. In the diagram, a concentration-visual range product of 0.3 ppm-km NO_2 corresponds to a colour shift which should be detectable in a polluted layer viewed against a relatively clean sky (EPA, 1982). At a visual range of 10 kilometres, 564 $\mu g/m^3$ (0.03 ppm) NO_2 might be required to colour the horizon noticeably, while at a visual range of 50 kilometers, 11 $\mu g/m^3$ (0.006 ppm) might be sufficient. However, the U.S., EPA (1982) cautions that calculation of human perception of NO_2 is not fully developed and that experimental observations are needed to evaluate the effect. In four examples, particle optical properties were shown to dominate NO_2 absorption in producing coloured hazes in Denver and Los Angeles (Charlson *et al.* 1972; Husar and White, 1976; Waggoner, 1977; Groblicki *et al.*, 1981). Thus, particle optical effects are alone capable of imparting a reddish-brown colour to a haze layer when viewed in backward scatter (sun in back of the observer), although sufficient NO_2 could increase the degree of colouration (Charlson *et al.* 1978). When the sun is in front of the observer, forward scatter by particles tend to wash out the trasmitted brownish light, i.e. particles diminish haze colouration.

FIGURE 4.6-2 Relative horizon brightness for selected values of the concentration-visual product, assuming b =3/(visual range). (Adapted from Hodkinson, 1966)



4.6.3 Particle Products of Photochemical Reaction

In northwestern Europe, northeastern United States, southern Canada, and western Canada, California, Tokyo, and Sydney, visibility reduction is an important effect of photochemical pollution episodes (OECD, 1979; Vickers and Munn, 1977; Harrison and Mathai, 1980). Photochemical reactions lead to the oxidation of sulphur dioxide and nitrogen dioxide to aerosol species which cause dense summer hazes over large areas. The aerosol in polluted photochemical air masses has been observed to reduce solar radiation to one-tenth of its incident value (OECD, 1979). In California, a close relation between visibility reduction and photochemical smog was observed when b_{sp} was measured during periods when ozone concentration was at its maximum (Grosjean and Friedlander, 1975).

The secondary particle products of photochemical reaction include sulphates, nitrates, and organic particulates. Soot or elemental carbon (a primary pollutant) and its effects on visibility will be mentioned in this section, but receives more extensive treatment in Section 4.6.4 on diesel exhaust particulates. The important causes of visibility reduction in conditions conducive to photochemical reaction include man-made sources of sulphur oxides, nitrogen oxides, volatile organics, while soot and other particles also play an important role in all atmospheric conditions (EPA, 1979). The following discussions in this section are largely adapted from the U.S. criteria document on particulates (1982) and the Canadian criteria document (NRCC, 1982) supplemented by more recent information on the chemical composition of particles contributing to visibility impairment and potential effects on fog formation.

Particles below 0.1 μm are sufficiently small compared with the wavelength of light to obey approximately the same laws of light scattering as do gas molecules (so-called Rayleigh or blue sky scattering) and have an inconsequential effect on visibility. Particles larger than about 2.5 μm intercept or scatter light in proportion to their cross-sectional area. Particles in the intermediate size range are of the same dimension as visible wavelengths. As a result of interference phenomena, this is the particle size range which is most effective in light scattering and thus may have a major effect on visibility.

Natural soil particulates emissions in the prairie provinces have been shown to be significantly greater than amounts emitted in other provinces (EAG, 1982). Since soil eroded particles have an average size range of 5 to over 100 µm, they would not contribute to the fine particulate fraction. LaDochy and Annett (1982) do indicate that slightly higher total particulate levels in Alberta, Saskatchewan, and Winnipeg are associated with soil erosion during drought conditions. However, Harrison and Mathai (1980) showed that visibility in Calgary is related to fine particle scattering.

The bulk of the population of particles below 2 µm arise by condensation from a vapour phase followed by coagulation. In most cities, the predominant aerosol that obscures visibility is a mixture of sulphate, organic compounds, and in some western U.S. cities, nitrate, all in the fine particle size range (Wolff et al., 1982).

Humidity has a very important effect on visibility because of the presence of hygroscopic particles such as sulphates and nitrates which have been particularly cited in this respect. Hygroscopic particles tend to absorb atmospheric water and thus increase the amount of light scattering. Tuomi (1975) developed relationships for the growth of particle size as a function of relative humidity, and has shown that when relative humidity rises from 40% to 80%, particle size will increase 40%. In addition, the imaginary index of refraction decreases because of the absorbed water. This combination of factors and results causes increased scattering. Covert et al.'s (1980) measurements suggest that extinction due to relative humidity will increase by a factor of two as relative humidity increases from 70% to 90%.

The relationship between dry particle scattering extinction and fine particle mass concentration (FMC) is sufficiently stable over a wide range of areas that reasonable quantitative estimates of visibility can be made with the Koschmeider relationship (cf. Section 4.6.1) where relative humidity is lower than 70% (MOI, 1983). Waggoner and Weiss (1980) showed that for various sites in the western United States, that $b_{sp}/FMC = 3.1\ m^2/g$ with a correlation coefficient of 0.95. The relatively high correlation suggests (assuming K=3.9 and 0.02 contrast threshold) that:

$$V = \frac{1250}{FMC} \qquad (5)$$

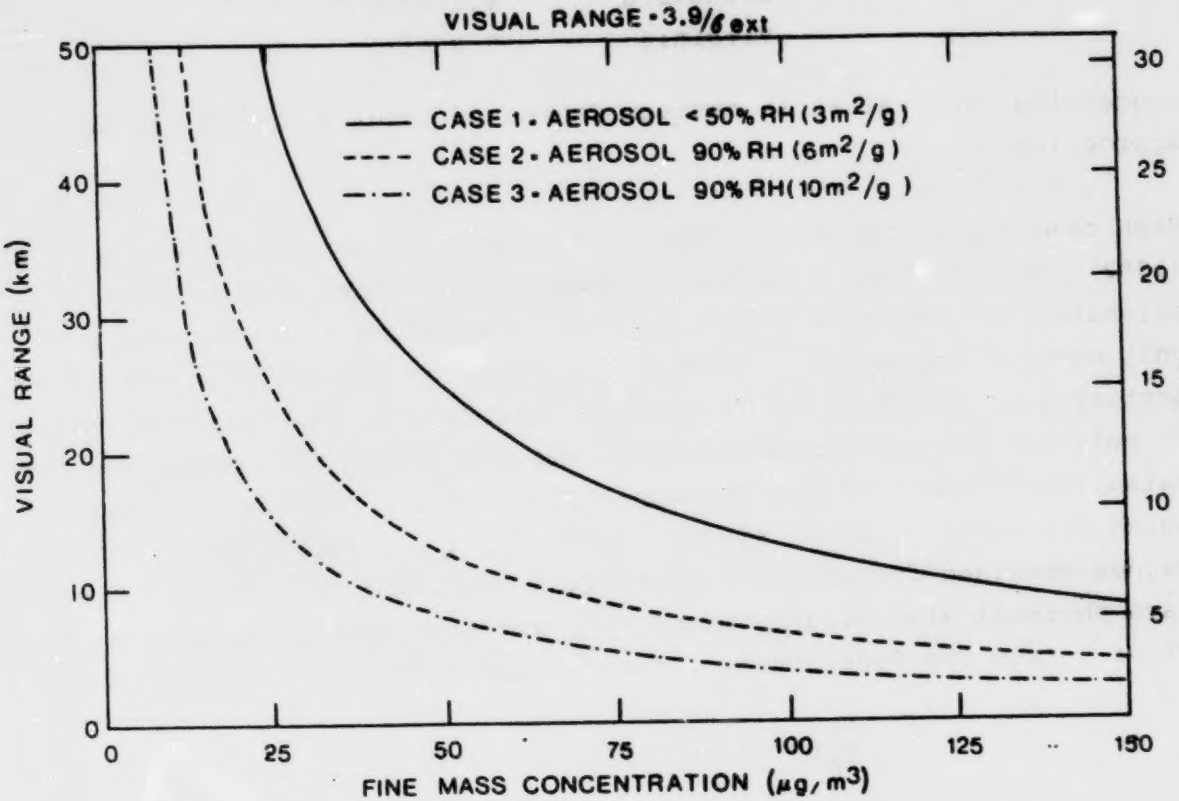
where visibility (V) is in kilometre units, and FMC in units of $\mu g/m^3$. This relation holds only for conditions of relative humidity less than 70% and where b_{ap}, particle absorption, is minimal (typical of non-urban areas; Waggoner and Charlson, 1976). The plotted values in Figure 4.6-3 show that the impact of a given increase in FMC is greater at high visibilities than at low ones. Thus, it is harder to maintain visibility in a high visibility area than to prevent noticeable change in visibility in an area of high particle concentration.

Figure 4.6-3 also illustrates results of the extension of the Koschmeider relationship considering relative humidity (RH) and particle absorption. The U.S. EPA criteria document (1982) indicates that to correct for the humidity effect the scattering efficiency, $\gamma = (b_{ap} + b_{sp})/FMC$, should be increased by a factor of 1.5 at 80% RH, and about 2 at 90% RH. The effects of humidity and particle absorption on visibility are significant for a given particle concentration. For example, when the fine mass concentration is 25 $\mu g/m^3$ with RH <50% (Case 1), visual range is about 50 km; at 90% RH (Case 2), this drops to about 25 km; and when there is high particle absorption (Case 3) and high RH, visual range is about 15 km.

Trijonis (1982a) has estimated the natural background levels of visibility and fine particles in rural eastern United States. Under natural background conditions, he estimates that there is a fine aerosol concentration of 5½ ± 2½ $\mu g/m^3$. The largest components would be organics (2 ± 2 $\mu g/m^3$) and water (1½ ± 1 $\mu g/m^3$), sulphates (about ½ $\mu g/m^3$) resulting in an average visual range of about 100 ± 50 km.

Representative values of measured scattering efficiency for U.S. urban areas are (Wolff et al.,1982):

FIGURE 4.6-3 Visual range as a function of fine mass concentration (determined from equilibrated filter) and Y, assuming the "standard" K=3.9. Because K is commonly lower in nonideal application, results from this relationship should not be compared directly to airport visibility data.



Reproduced from MOI, 1983

CASE 1. $\gamma = 3\ m^2/g$; representative of a dry aerosol, (USEPA 1981) at $\leq 50\%_2$RH. Absorption may be 10% of extinction where σ_{sp}/unit mass = 2.7 m^2/g. This is close to typical measurements in western areas but below most eastern data (USEPA 1981).

CASE 2. $\gamma = 6\ m^2/g$; representative of the same aerosol as in 1) at 90% humidity, σ_{sp} increased by a factor of 2.

CASE 3. $\gamma = 10\ m^2/g$; representative of the similar aerosol, but with absorption accounting for 40% of extinction. Such high absorption (predominantly associated with carbon) is likely only in urban areas.

Detroit	$4.8\ m^2/g$
Denver	$3.1\ m^2/g$
Louisiana	$4.7\ m^2/g$
Virginia	$5.8\ m^2/g$

suggesting that values in eastern Canada of about $4.5\ m^2/g$ may be appropriate.

Each case may be representative of a variety of aerosols. Wolff et al. (1982) conclude from a series of regional and urban studies that sulphates are the most efficient light scattering fine particle per unit mass of dry weight. Thus, the scattering efficiency of dry fine particles is not constant because it depends upon the relative amount of sulphate. Using regression analyses, Wolff and his co-workers have established relationships between extinction and chemical composition which are given in Table 4.6-1. Wolff et al. (1982) suggest that the values obtained for specific scattering coefficients (for each species) have physical significance since they are essentially the same at all sites. From the four studies, they obtained the dry particle relation

$$\frac{b_{sp}}{FMC} = 7.32\ (\%S/100) + 2.26$$

where %S refers to the portion of the total fine particle mass that is sulphur. In Detroit, the contributors to the observed light extinction were: sulphate and its associated water, 65%; carbon, 20%; Rayleigh scattering, 7%; NO_2, 4%; and other fine particulate species, 4%. However, Denver's haze included significant contributions from nitrates as well. In Denver, most of the nitrate was in the fine particle mass fraction, and accounted for 30% of the observed light extinction, while in Detroit, the majority was in the coarse particle fraction and contributed negligibly to the extinction budget. Organic fine particulates and elemental carbon also are important contributors to the extinction budget. In the Detroit study (Wolff et al., 1982) 20 percent of the observed light extinction was from carbon, the majority of which was organic compounds. In the rural eastern United States, light scattering by carbon accounted for an average of 13% of total extinction (Ferman et al., 1981). Again most of the carbon (80%) was organic with some

TABLE 4.6-1

COMPARISON OF THE REGRESSION COEFFICIENTS AND STANDARD ERROR AT 4 SITES

All values are in m^2/g

Site	Simple Coefficient FPM	Multiple regression coefficients SO_4^-	NO_3^-	EC	OC	TC	R	$SO_4^-/(1-RH)$	$NO_3^-/(1-RH)$
Denver	3.07	8.1 ± 0.6	3.2 ± 0.4	2.8 ± 1.2	4.7 ± 1.1		1.5 ± 0.3	2.1 ± 0.2	1.7 ± 0.2
Louisiana	4.73	8.8 ± 0.6	a	b	b	5.0 ± 1.5	1.6 ± 0.6	c	c
Virginia	5.80	7.4 ± 0.6	a	b	b	5.6 ± 2.8	1.8 ± 0.8	d	a
Detroit	4.80	8.5 ± 0.5	a	b	b	3.6 ± 1.6	1.7 ± 1.0	3.2 ± 0.3	a

a - Values too low to include in regression
b - EC and OC combined in TC because EC values near lower limit of detection
c - No h_{sw} measurements
d - Could not be determined

EC - elemental carbon
OC - organic carbon
TC - total carbon

Source: Wolff et al., 1982

relationships:

$$bext = A\,SO_4 + BNO_3 + cEC + dOC + eR$$

or in special cases where sulphate is present primarily as $(NH_4)_2SO_4$

$$bext = \frac{aSO_4}{1-RH} + \frac{bNO_3}{1-RH} + c\,EC + d\,OC + e\,R$$

indication of being derived from natural sources. Organic particles formed in photochemical smog have been studied and it has been found that oxygenated organic materials are important ingredients in the size range between 0.1 and 2 μm (Schuetzle et al., 1975). The elemental carbon portion is addressed in the following section on diesel exhaust particulates.

Condensation or particle growth due to water absorption begins at low relative humidities (<70%) as previously indicated. As relative humidity increases to 90%, the initial haze droplets can grow to become fog droplets with a resulting decrease in visibility (Petterssen, 1958; cf. Figure 4.6-3). Thick "London type" fogs or smogs can be generated at relative humidities less than 100 percent if there is a high concentration of hygroscopic particles which serve as efficient condensation nuclei (NRCC, 1982).

Hung and Liaw (1981) indicate that the condensation nuclei (particles with diameter equal to or greater than 0.1 μm) associated with a polluted atmosphere provides more favourable conditions to produce dense fog than condensation nuclei associated with a clean atmosphere. They also noted that condensation nuclei with high hygroscopicity (e.g. sulphate, nitrate) provide more superior conditions for the formation of dense fog than condensation nuclei with lower hygroscopicity. The visibility in the fog will depend upon the concentration and size of the resulting droplets. These fogs may also be highly acidic (Wisniewski, 1982; Hileman, 1983).

4.6.4 Diesel Exhaust Particles

Emissions of soot (elemental carbon) from light duty diesel vehicles are of potential significance to visibility as indicated in the previous section. Approximately 70 to 90% of the particulate matter emitted from light duty diesels is soot (Amann and Siegla, 1982). Soot strongly absorbs light in addition to scattering light; Trijonis (1982b) indicates that fine soot particles have a light extinction efficiency of 3 to 4 times that of other fine particles. The extinction coefficient for fine elemental carbon is 12 ± 3 m^2/g (Waggoner and Weiss, 1980; Groblicher et al., 1981) with 9 m^2/g absorption and 3 m^2/g scattering (compared with 6-7 m^2/g for dry sulphate cf. Section 4.6.3).

The effect on carbon particle absorption on light extinction has been measured in U.S. urban and rural areas (Weiss and Waggoner, 1981). In five urban areas, the absorption ranged from 0.35 to 0.50 of total extinction and in six rural areas, it varied from 0.13 to 0.27. The dramatic effect on visual range by light absorption by carbon is illustrated as Case 3 in Figure 4.6-3. The scattering portion of light extinction due to elemental carbon has been measured to range from 6% during summer to 35-36% during winter in various U.S. cities (Wolff et al. 1981).

Trijonis (1982b) investigated the impact of light duty diesels on California visibility for a future scenario of 20% "dieselization' of the automobile fleet. His haze budget calculations indicate that increased soot emissions by diesels would possibly reduce visibility about 10 to 25%. Another potential side effect of increased elemental carbon concentrations is the possible increase in SO_4 loadings because soot is suspected to be a major catalyst for SO_4 formation (Wolff, 1981; Chang and Novakov, 1981). Sulphate has been demonstrated to be one of the dominant light scattering species.

4.6.5 Summary

Visibility is recognized as being important to perceived air quality by people in general and by government agencies. One of the criteria used to establish the Canadian maximum air quality objective for suspended particulates was protection against visibility impairment. Recently both the U.S. EPA (1982) and the Canadian NRC (1982) have recommended that standards (objectives) be established for fine particles with visibility as a criteria on the basis of the relatively well defined relationship between visual range and fine particle mass.

The basis for estimating visual range from fine particle mass concentration has been provided in Section 4.6.3. The importance of sulphate in visibility reduction is firmly established, while nitrates, organic compounds, carbon (soot) are more site dependant. The semi-empirical relationships developed by Wolff et al. (1982) may be used as an indicator of the relative importance of the various chemical species present in the fine particle mode to visibility impairment.

The Memorandum of Intent document (MOI, 1983) concluded that "available data suggest that nitrates exist predominantly in the vapour phase and are for the most part of little consequence to visibility in eastern North America." Production of aerosol nitrate instead of nitric acid vapour may require neutralization by ambient ammonia (NAS, 1977). In the western United States particulate nitrate has been shown to be of substantial importance in visibility reduction on a local scale (Trijonis, 1982; Groblicki et al, 1981).

The contribution of nitrogen dioxide to reduction of visual range is small, but it may cause a brownish haze in some circumstances; however, in the majority of cases, brownish haze has been attributed to particle scattering.

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Section 3. AIR QUALITY CRITERIA AND STANDARDS

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Section 4.3 TERRESTRIAL EVIRONMENT

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GLOSSARY

GLOSSARY

Abscission: The process whereby leaves, leaflets, fruits, or other plant parts become detached from the plant.

Acid: A substance that can donate hydrogen ions.

Acute toxicity: Any poisionous effect produced by a single short-term exposure, that results in severe biological harm or death.

Acid rain: Rain having a pH less than 5.6, the minimum expected from atmospheric CO_2.

Acute respiratory disease: Respiratory infection, usually with rapid onset and of short duration.

Adsorption: The adhesion of molecules in an extremely thin layer to the surfaces of solids or liquids with which they are in contact.

Air pollution: A substance present in the ambient atmosphere, resulting from the activity of man or from natural processes, which may cause damage to human health or welfare, the natural environment, or materials or objects.

Alkali: A salt of sodium or potassium capable of neutralizing acids.

Alveolar: Pertaining to the alveoli or small air pockets of the lungs.

Alveolar macrophages (AM): Larger mononuclear, phagocytic cells found on the alveolar surface, reponsible for the sterility of the lung.

Alveolar oxygen partial pressure (PAO_2): Partial pressure of oxygen in the air contained in the air sacs of the lungs.

Ambient air: The surrounding, well-mixed air.

Anaemia: A reduction below normal in the number of erythrocytes (red blood cells) per cubic millimeter, in the quantity of hemoglobin, or in the volume of packed red cells per 100 milliliters of blood.

Anaerobic: Living, active or occurring in the absence of free oxygen.

Angina pectoris: Severe constricting pain in the chest which may be caused by depletion of oxygen delivery to the heart muscle; usually caused by coronary disease.

Angiosperm: A plant having seeds enclosed in an ovary; a flowering plant.

Anoxia: Absence or lack of oxygen; reduction of oxygen in body tissues below physiologic levels.

Anthropogenic: Relating to the impact of man and his activities on the natural world.

Aromatic: Belonging to that series of carbon-hydrogen compounds in which the carbon atoms form closed rings containing unsaturated bonds (as in benzene).

Asthma: A term currently used in the context of bronchial asthma in which there is widespread narrowing of the airways of the lung. It may be aggravated by inhalation of pollutants and lead to "wheezing" and shortness of breath.

Atheromatous: Pertaining to fatty degeneration or thickening of the wall of the larger arteries.

Atmosphere: The body of air surrounding the earth. Also, a measure of pressure (atm.) equal to the pressure of air at sea level, 14.7 pound per square inch.

Atmospheric deposition: Removal of pollutants from the atmosphere onto land, vegetation, water bodies or other objects, be aborption, sedimentation, Brownian diffusion, impaction, or precipitation in rain.

Background measurement: A measurement of pollutants in ambient air due to natural sources; usually taken in remote areas.

Benthos: Organisms living on or at the bottom of a body of water.

Biomass: That part of a given habitat consisting of living matter.

Bronchi: The first subdivisions of the trachea which conduct air to and from the bronchioles of the lungs.

Bronchiole: One of the finer subdivisions of the bronchial (trachea) tubes, less than 1 mm in diameter, and having no cartilage in its wall.

Bronchitis: Inflammation of the mucous membrane of the bronchial tubes. It may aggravate an existing asthmatic condition.

Bronchoconstrictor: An agent that causes a reduction in the caliber (diameter) of a bronchial tube.

Carbon monoxide: An odourless, colourless, toxic gas with a strong affinity for haemoglobin and cytochrome; it reduces oxygen absorption capacity, transport and utilization.

Carboxyhaemoglobin: A fairly stable union of carbon monoxide with haemoglobin which interferes with the normal transfer of carbon dioxide and oxygen during circulation of blood. Increasing levels of carboxyhaemoglobin results in various degrees of asphyxiation, including death.

Carcinogen: Any agent producing or playing a stimulatory role in the formation of a malignancy.

Carinogenesis: The development of a carcinoma, a malignant new growth of epithelial cells.

Cardiac output: The volume of blood passing through the heart per unit time.

Cardiovascular: Relating to the heart and the blood vessels or the circulation.

Cation: A positively charged ion.

Cellulose: The basic substance which is contained in all vegetable fibres and in certain man-made fibres. It is a carbohydrate and constitutes the major substance in plant life. Used to make cellulose acetate and rayon.

Cellulose acetate: Commonly refers to fibres or fabrics in which the cellulose is only partially acetylated with acetate groups. An ester made by reacting cellulose with acetic anhydride with SO_4 as a catalyst.

Cellulose rayon: A generated cellulose which is chemically the same as cellulose except for physical differences in molecular weight and crystallinity.

Central nervous system (CNS): Brain and spinal cord together.

Chamber study: Research conducted using a closed vessel in which pollutants are reacted or substances exposed to pollutants.

Chlorosis: Discolouration of normally green plant parts that can be caused by disease, lack of nutrients, or various air pollutants, resulting in the failure of chlorophyll to develop.

Chromosome: One of the bodies (46 in man) in the cell nucleus that is the bearer and carrier of genetic information.

Chromatid: Each of the two strands formed by longitudinal duplication of a chromosome that becomes visible during an early stage of cell division.

Chronic Obstructive Lung (Pulmonary) Disease: Disease process which causes decreased ability of the lungs to perform their function of ventilation e.g. chronic bronchitis, pulmonary emphysema, chronic asthma, chronic bronchiolitis.

Chronic respiratory disease (CRD): A persistent or long-lasting intermittent disease of the respiratory tract.

Cilia: Motile, often hairlike extensions of a cell surface.

Ciliary action: Movements or cilia in the upper respiratory tract, which moves mucous and foreign material upward.

Claudication: Limping or lameness; a complex of symptoms frequently associated with occlusive arterial diseases of the limbs.

Coefficient of haze (COH): A measurement of visibility interference in the atmosphere.

Community exposure: A situation in which people in a sizeable area are subjected to ambient pollutant concentrations.

Conifer: A plant, generally evergreen, needle-leafed, bearing naked seeds singly or in cones.

Coronary: Pertaining to the arteries and veins of the heart.

Corrosion: Destruction or deterioration of a material because of reaction with its environment.

Crevice corrosion: Localized corrosion occurring within crevices on metal surfaces exposed to corrosives.

Cultivar: An organism produced by parents belonging to different species or to different strains of the same species, originating and persisting under cultivation.

Cytochrome: A class of haemoprotein whose principal biological function is electron and/or hydrogen transport.

Leukemia: A usually fatal cancerous disease characterized by excessive production of white blood cells.

Pancytopenia: A reduction in all cellular elements of the blood.

Dark adaptation: The process by which the eye adjusts under reduced illumination and the sensitivity of the eye to light is greatly increased.

Deciduous plants: Plants which drop their leaves at the end of the growing season.

Degradation (textiles): The decomposition of fabric or its components or characteristics (colour, strength, elasticity) by means of light, heat, or air pollution.

Detritus: Loose material that results directly from disintegration.

Diffusion: The process by which particles of gases, liquids, or solids intermingle as a result of their spontaneous movement caused by thermal agitation, and move from a region of higher concentration to a region of lower concentration.

DL_{CO}: The diffusing capacity of the lungs for carbon monoxide. The ability of the lungs to transfer carbon monoxide from the alveolar air into the pulmonary capillary blood.

Dose: The quantity of a substance to be taken all at one time or in fractional amounts within a given period; also the total amount of a pollutant delivered or concentration per unit time times time.

Dose-response curve: A curve on a graph based on responses occurring in a system as a result of a series of stimuli intensities or doses.

Double blind technique: A method of investigation in which neither the subject nor the investigator working with the subject or data knows what treatment, if any, the subject is receiving.

Dry deposition: The processes by which matter is transferred to ground from the atmosphere, other than precipitation; includes surface absorption of gases and sedimentation, Brownian diffusion and impaction of particles.

Dyspnea: Shortness of breath; difficulty or distress in breathing; rapid breathing.

Ecosystem: The interacting system of a biological community and its environment.

Edema: Pressure of excess fluid in cells, intercellular tissues or cavities of the body.

Elastomer: A synthetic rubber product which has the physical properties of natural rubber.

Electrocaridogram (EKG): A tracing made by an electrocardiograph which measures changes of electrical potential occurring during the heartbeat.

Emphysema: An anatomic alteration of the lung, characterized by abnormal enlargement of air spaces distal to the terminal bronchioles, due to dilation or destructive changes in the alveolar walls.

Endogenous: Originating within the organism.

Epidemiology: The study of the relationships of the various factors determining the frequency and distribution of diseases in a human community.

Epilimnion: The uniformly warm upper layer of a lake when it is thermally stratified in summer. The layer above the metalimnion.

Erosion corrosion: Acceleration or increase in rate of deterioration or attack on a metal because of relative movement between a corrosive fluid and the metal surface. Characterized by grooves, gullies, or waves in the metal surface.

Ethanol: Ethyl alcohol.

Eutrophication: Elevation of the level of nutrients in a body of water, which can contribute to accelerated plant growth and filling.

Expiratory (maximum) flow rate: The maximum rate at which air can be expelled from the lungs.

Exposure level: Concentration of a contaminant to which an individual or a population is exposed.

Fibre-reactive dye: A water-soluble dyestuff which reacts chemically with the cellulose in fibres under alkaline conditions; the dye contains two chlorine atoms which combine with the hydroxyl groups of the cellulose.

Fibrillation (cardiac): Rapid, irregular contractions of the muscle fibres of the heart.

Forced expiratory flow (FEF): The rate at which air can be expelled from the lungs; see expiratory flow rate.

Forced expiratory flow, 25% - 75% (FEF$_{25\%-75\%}$). The mean rate of expiratory gas flow between 25 and 75 percent of the forced expiratory vital capacity.

Forced expiratory volume (FEV): The maximum volume of air that can be expired in a specific time interval when starting from maximal inspiration.

Forced vital capacity (FVC): The greatest volume of air that can be exhaled from the lungs under forced conditions after a maximum inspiration.

Functional residual capacity: The volume of gas remaining in the lungs at the end of a normal expiration. It is the sum of expiratory reserve volume and residual volume.

Gas exchange: Movement of oxygen from the alveoli into the pulmonary capillary blood as carbon dioxide enters the alveoli from the blood.

Globin: The protein constituent of haemoglobin.

Haemoglobin (Hb): The red, respiratory protein of the red blood cells, haemoglobin transports oxygen from the lungs to the tissues as oxyhaemoglobin (HbO_2) and returns carbon dioxide to the lungs as haemoglobin carbamate, completing the respiratory cycle.

Haldane constant: Ratio of the stability constant for carboxyhaemoglobin to that for oxyhaemoglobin; a measure of the relative affinity of haemoglobin for carbon monoxide as compared to its affinity for oxygen.

Herbivore: A plant-eating animal.

Hydrocarbons: A vast family of compounds containing carbon and hydrogen in various combinations; found especially in fossil fuels. Some contribute to photochemical smog.

Hydroxyl radical: Unstable, electrically neutral fragment of a molecule containing one oxygen atom and one hydrogen atom. It is formed by disruption of a water (or other hydroxyl-containing) molecule, as a result of exposure to far ultraviolet light or other high-energy radiation such as X-rays.

Hygroscopic: Pertaining to a marked ability to accelerate the condensation of water vapor.

Hyperplasia: Increase in the number of cells in a tissue or organ excluding tumor formation.

Hypolimnia: Portions of a lake below the thermocline, in which water is stagnant and uniform in temperature.

Hypoxemia: Deficient oxygenation of the blood; hypoxia.

Hypoxia: Low oxygen content or tension. Anaemia hypoxia is due to reduction of the oxygen-carrying capacity of the blood as a result of a decrease in the total haemoglobin or an alteration of the haemoglobin constituents.

Infarction: Sudden insufficiency of arterial or venous blood supply due to emboli, thrombi, or pressure.

Interstitial pneumonitis: A chronic inflammation of the interstitial tissue of the lung, resulting in compression of air cells.

In vitro: Outside the living organism.

In vivo: Within the living organism.

Ischemia: Local anaemia due to mechanical obstruction (mainly arterial narrowing) of the blood supply.

Isopleth: On a map, a line connecting points at which a particular variable has a specified constant value.

Lesion: A wound, injury or other more or less circumscribed pathologic change in the tissues.

Leukocyte: Any of the white blood cells.

Lipids: A heterogeneous group of substances including fats, waxes, phosphatides cerebrosides and related or derived compounds which occur widely in biological materials. They are characterized as a group by their extractability in nonpolar organic solvents.

Lymphocytes: White blood cells formed in lymphoid tissue throughout the body, they comprise about 22 to 28 percent of the total number of leukocytes in the circulating blood and function in immunity.

Macrophage: Any large, ameboid, phagocytic cell having a nucleus without many lobes, regardless or origin.

Mean (arithmetic): The sum of observations divided by sample size.

Median: A value in a collection of data values which is exceeded in magnitude by one-half the entries in the collection.

Mesophyll: Thin-walled cells in the interior of a leaf; rich in chloroplasts.

Metastasis: The shifting of a disease from one part of the body to another; the appearance of neoplasms in parts of the body remote from the seat of the primary tumor.

Methylene chloride (Dichloromethane; CH_2Cl_2): A compound which causes an elevation of carboxyhaemoglobin; a commercial solvent.

Microequivalents per litre (µeq/L): A unit of concentration measuring relative acidity.

Milligrams per cubic meter (mg/m^3): A measure of concentration of a substance. For example, the weight in milligrams of CO contained in one cubic meter of the ambient air, which may be converted to "parts per million" at one atmosphere by multiplication by the factor 0.873 at 25°C, or by the factor 0.800 at 0°C. At pressures other than one atmosphere (760 torr) such a factor should be multiplied by an additional factor of 760/p, where p is the ambient pressure in torr.

Mitochondria: Organelles of the cell cytoplasm which contain enzymes active in the conservation of energy obtained in the aerobic part of the breakdown of carbohydrates and fats, in process called respiration.

Mobile sources: Automobiles, trucks and other pollution sources which are not fixed in one location.

Morphological: Relating to the form and structure of an organism or any of its parts.

Mucociliary clearance: Removal of materials from the upper respiratory tract via ciliary action.

Mutagenic: Having the power to cause mutations. A mutation is a change in the character of a gene (a sequence of base pairs in DNA) that is perpetuated in subsequent divisions of the cell in which it occurs.

Myocardial infarction: A necrotic (dead) area of the heart muscle, usually as a result of occlusion of a coronary artery.

Myocardium: Muscle of the heart.

N/P Ratio: Ratio of nitrogen to phosphrous dissolved in lake water, important due to its effect on plant growth.

Necrosis: Death of cells that can discolour areas of a plant or kill the entire plant.

Necrotic: Pertaining to the pathologic death of one or more cells, or of a portion of tissue or organ, resulting from irreversible damage.

Neonate: A newborn.

Nitrate: A salt or ester of nitric acid (NO_3^-).

Nitrogen oxide: A compound composed of only nitrogen and oxygen. Components of photochemical smog.

Nitrosamine: A compound consisting of a nitrosyl group connected to the nitrogen of an amine.

Oligotrophic: A body of water deficient in plant nutrients; also generally having abundant dissolved oxygen and no marked stratification.

Oxidant: A chemical compound which has the ability to remove electrons from another chemical species, thereby oxidizing it; also, a substance containing oxygen which reacts in air to produce a new substance, or one formed by the action of sunlight on oxides of nitrogen and hydrocarbons.

Oxyhaemoglobin: Haemoglobin in combination with oxygen. It is the form of haemoglobin present in arterial blood.

Ozone layer: A layer of the stratosphere from 20 to 50 km above the earth's surface characterized by high ozone content produced by ultraviolet radiation.

Ozone scavenging: Removal of O_3 from ambient air or plumes by reaction with NO, producing NO_2 and O_2.

Particulates: Fine liquid or solid particles such as dust, smoke, mist, fumes or smog, found in the air or in emissions.

Parts per million (ppm): A measure to concentration of a substance. For example, the volume in liters of CO contained in 1,000,000 liters of the ambient air, which may be converted to "milligrams per cubic meter" by multiplication by the factor 1.145 at 25°C, or by the factor 1.250 at 0°C. At pressures other than one atmosphere (760 torr) such a factor should be multiplied by an additional factor of p/760, where p is the ambient pressure in torr. Parts per hundred million: pphm.

Pathogen: Any virus, microorganism, or other substance causing disease.

Pathology: Study of the structural and functional changes produced by diseases, e.g. abnormalities.

Pathophysiological: Derangement of function seen in disease; alteration in function as distinguished from structural defects.

Peribronchiolitis: Inflammation of area around the bronchioles.

Perinatal: Occurring in the period preceding during, or after birth.

Permanent-press fabrics: Fabrics in which applied resins contribute to the easy care and appearance of the fabric and to the crease and seam flatness by reacting with the cellulose on pressing after garment manufacture.

Peroxyacetyl nitrate (PAN): Pollution created by action of sunlight on hydrocarbons and NO_x in the air; an ingredient of photochemical smog.

pH: A measure of the acidity or alkalinity of a material, liquid, or solid. pH is represented on a scale of 0 to 14 with 7 being a neutral state, 0 most acid, and 14 most alkaline.

Phagocytosis: Ingestion, by cells such as macrophages, of other cells, bacteria, foreign particles, etc.; the cell membrane engulfs solid or liquid particles which are drawn into the cytoplasm and digested.

Phlegm: Viscid mucous secreted in abnormal quantity in the respiratory passages.

Photochemical oxidants: Primary ozone, NO_2, PAN with lesser amounts of other compounds formed as products of atmospheric reactions involving organic pollutants, nitrogen oxides, oxygen, and sunlight.

Photochemical smog: Air pollution caused by chemical reaction of various airborne chemicals in sunlight.

Photolysis: Decomposition upon irradiation by sunlight.

Photosynthesis: The process in which green parts of plants, when exposed to light under suitable conditions of temperature and water supply, produce carbohydrates using atmospheric carbon dioxide and releasing oxygen.

Phytoplankton: Minute aquatic plant life.

Phytotoxic: Poisonous to plants.

Planktonic communities: Community of microorganisms, consisting of plants (phytoplankton) and animals (zooplankton), inhabiting open-water regions of lakes and rivers.

Plume: Emission from a flue or chimney, usually distributed streamlike downwind of the source, which can be distinguished from the surrounding air by appearance or chemical characteristics.

Point source: A single stationary location of pollutant discharge.

Precipitation: Any of the various forms of water particles that fall from the atmosphere to the ground, rain, snow, etc.

Precursor: A substance from which another substance is formed; specifically, one of the anthropogenic or natural emissions or atmospheric constituents which reacts under sunlight to form secondary pollutants comprising photochemical smog.

Psychomotor: Pertaining to motor effects of cerebral or psychic activity.

Pulmonary: Relating to the lungs.

Pulmonary edema: An accumulation of excessive amounts of fluid in the lungs.

Pulmonary resistance: Sum of airway resistance and viscous tissue resistance.

Radicals: Unstable fragments of molecules which have an unpaired electron and tend to react or change rapidly into more stable substances.

Rayleigh scattering: Coherent scattering in which the intensity of the light of wavelength λ, scattered in any direction making an angle with the incident direction, is directly proportional to $1 + \cos^2 \theta$ and inversely proportional to λ^4.

Reactive dyes: Dyes which react chemically with cellulose in fibres under alkaline conditions. Also called fiber reactive or chemically reactive dyes.

Residual capacity: The volume of air remaining in the lungs after a maximum expiratory effort; same as residual volume.

Residual volume (RV): The volume of air remaining in the lungs after a maximal expiration. RV = TLC - VC.

Rotifer: One of a division of many-celled, microscopic, aquatic organisms usually found in stagnant fresh water.

Sequela (ae): A lesion or affection following or caused by an attack of disease.

Stomatal: Pertaining to the minute opening on the surface of a leaf, surrounded by a pair of guard cells that regulate the size of the opening.

Stratosphere: An upper region of the earth's atmosphere, above about 10 to 16 kilometres, in which clouds are rare and there is little change of temperature with altitude.

Sink: An absorber of a substance, or a process which acts as a removal or dissipation mechanism.

Stress corrosion cracking: Cracking caused by simultaneous presence of tensile stress and a specific corrosive medium. The metal or alloy is virturally unattached over most of its surface, while fine cracks progress through it.

Sulphur dioxide (SO_2): Colourless gas with pungent odour released primarily from burning of fossil fuels, such as coal, containing sulphur.

Synergistic: A relationship in which the combined action or effect of two or more components is greater than that of the components acting separately.

Teratogenesis: The disturbed growth processes resulting in a deformed fetus.

Teratogenic: Causing or relating to abnormal development of the fetus.

Threshold: The level at which a physiological or psychological effect begins to be produced.

Threshold limit valve (TLV): Airborne concentration of a substance representing occupational conditions under which the American Conference of Governmental Industrial Hygenists believes that nearly all workers may be repeatedly exposed day after day without adverse effect.

Threshold limit valve - time weighted average (TLV-TWA): The time-weighted average concentration of a substance in a workplace for a normal 8-hour workday and a 40-hour workweek, to which nearly all workers may be repeatedly exposed, day after day, without adverse effect.

Thyrotoxicosis: Toxic condition due to hyperactivity of the thyroid gland.

Total lung capacity (TLC): The sum of all the compartments of the lung, or the volume of air in the lungs at maximum inspiration.

Total suspended particulates (TSP): Solid and liquid particles present in the atmosphere.

Trachea: Commonly known as the windpipe, a cartilaginous air tube extending from the larnyx (voice box) into the thorax (chest) where it divides, serving as the entrance to each of the lungs.

Transpiration: The process of loss of water vapour from plants.

Troposphere: The portion of the earth's atmosphere which extends from the surface out to an altitude of about 7 to 10 miles or 10 to 16 kilometers.

Vascular: Of or pertaining to the blood vessels.

Vasodilation: Dilation of a blood vessel, increasing the blood flow.

Vigilance: A stage of alertness requiring continuous attention over long periods of time.

Viscose rayon: Filaments of regenerated cellulose coagulated from a solution of cellulose xanthate. Raw materials can be cotton linters or chips of spruce, pine, or hemlock.

Visual range: The distance at which an object can be distinguished from background.

Vital capacity: The greatest volume of air that can be exhaled from the lungs after a maximum inspiration.

Wet deposition: The process by which atmospheric substances are returned to earth in the form of rain or other precipitation.

Zooplankton: Minute animal life floating or swimming weakly in a body of water.